UNITED STATES BANKRUPTCY COURT
FOR THE WESTERN DISTRICT OF NORTH CAROLINA
CHARLOTTE DIVISION

IN RE:	)	
	)	
GARLOCK SEALING TECHNOLOGIES	)	
LLC, et al,	)	No. 10-BK-31607
	)	
Debtors.	)	VOLUME II-B AFTERNOON SESSION
_____	)	

TRANSCRIPT OF ESTIMATION TRIAL
BEFORE THE HONORABLE GEORGE R. HODGES
UNITED STATES BANKRUPTCY JUDGE
JULY 23, 2013

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I N D E X

TUESDAY AFTERNOON, JULY 23, 2013

(Court called to order at 2:15 p.m.)

THE COURT: Start with the next witness.

MR. SCHACHTER: May I proceed, Your Honor?

THE COURT: Yes, sir.

MR. FINCH: Your Honor, one housekeeping matter before we begin. Defendants -- or Garlock has not been providing us with power points that it's going to use on its experts. I'm fine with that as long as the sauce rule is in effect and we don't have to give our power points. We asked about that last week and there was no agreement about that so I am perfectly fine to proceed on that basis.

MR. CASSADA: That's fine. I thought we did have an agreement on it, but that's absolutely fine.

THE COURT: Well, if you can work out one, great. If not we'll just do it the same, yes.

MR. SCHACHTER: Your Honor, we call Dr. Thomas Sporn to the stand.

THE COURT: Okay.

THOMAS SPORN,

being first duly sworn, was examined and testified as follows:

DIRECT EXAMINATION

BY MR. SCHACHTER:

Q. Good afternoon, Dr. Sporn. Would you please introduce yourself to the court.

A. Good afternoon, Mr. Schachter. My name is Dr. Thomas Sporn.
Q. And where do you practice medicine?
A. I practice medicine at Duke University Medical Center up the road in Durham, North Carolina.
Q. What is your position there, sir?
A. I am a pathologist. I'm an associate professor of pathology within the medical school and I'm an attending pathologist at the hospital medical center.
Q. On the slide we have here it says you're the head of the section of pulmonary and thoracic pathology at Duke University. What are your activities in that capacity?
A. Well, I'm a physician who's involved in direct patient care. I don't have my own patients. I don't -- people don't come to see me in clinic. But I'm in charge of -- the lead pathologist in issuing diagnoses at patients who come to Duke Hospital who have thoracic disease.

That can mean that I interpret small biopsy specimens. I can interpret small cytology specimens, a few cells that have come off the patient's chest wall into their airway, and as part of an evaluation for lung cancers. I examine whole organs. I examine patients who have died in our hospital with suspected thoracic disease. So I'm a physician who is in charge with the diagnostic end of taking care of folks with diseases of the chest.

Q. Are you board certified in any specialties?
A. I'm board certified in internal medicine. I'm also board certified in anatomic pathology and forensic pathology.
Q. How long have you been at Duke, sir?
A. Since 1993.
Q. Does this briefly summarize your education? If you can talk us through it in very brief terms. We're trying to be efficient.
A. Sure. In brief terms I never for the life of me thought I would end up as a pathologist. Very few people who go to medical school do. I started out my training with the expectation that I was going to be a treating doctor. And I've always had an interest in thoracic disease and trauma and critical care, and that's how I began my initial training after graduating from Georgetown School of Medicine.

I stayed on as a resident in internal medicine. I completed the fellowship in chest disease and critical care medicine.

And following a brief period out in private practice, I decided the science of medicine was more in line with my skill set and I returned to academics, to Duke where I studied pathology.

I was appointed medical examiner for the County of Durham here in North Carolina and later as one of the assistant state chief medical examiners down the road in Chapel Hill.

I returned to Duke to complete the remainder of my training, including a fellowship or period of advanced training in lung pathology under the auspices of Dr. Victor Roggli, and I've remained there ever since.

Q. Sir, there was a time when you mentioned that you were a treating physician for a brief period of time in private practice. When was that?

A. That was in 1992.

Q. Where was that?

A. That was out on the West Coast in Puget Sound in Bremerton, Washington.

Q. Did you have an occasion ever to treat people that had mesothelioma?

A. I did. The major employer in the town of Bremerton is the Puget Sound Naval Shipyard. At this point in our country's career we're not building so many battleships and aircraft carriers anymore as we are decommissioning and mothballing them. And a great many of the men and women who worked in the shipyard were decommissioning our old warships from the second world war and beyond, and a lot of them had asbestos-associated diseases. So in my patient population I had asbestotics, I had patients with pleural fibrosis, I had patients with mesothelioma.

Q. Did that have a role in your decision to pursue further education and training?

A. Yes. As a city kid from Washington, DC, I hadn't had much experience in that regard, and it became quite interesting to me.

For the brief time that I was out in Washington state I struck up a friendship with a very famous pulmonary pathologist out there who has a special interest in asbestos disease, Dr. Sam Hammar, and it was Sam that convinced me that pulmonary pathology would be a very attractive career goal for myself and he urged that I consider looking at Duke, amongst other places.

Q. And at Duke have you had a specialty in your research interests?

A. Well, I'm not a researcher in terms that I run a lab and work with animal models and gels and things like that. My research has been observational and reporting on diagnostic techniques and our experience in our own little division with asbestos disease and mesothelioma.

Q. Being at Duke, do you have an opportunity to encounter mesothelioma on a regular basis?

A. Yes, sir, very much so. I mean -- and that's due to the interest of the -- some of the treating oncologists and thoracic surgeons at my hospital. We see a tremendous amount of referral cases from the shipyard areas, both in South Carolina and Southern Virginia, as well as elsewhere in the country.

So I see -- I see a tremendous amount of mesotheliomas. I was working on one yesterday as I was preparing for this trial in terms of diagnosis and I was also examining a lung yesterday of a gentleman who had had a radical extrapleural pneumonectomy. So I see a tremendous amount of mesotheliomas just in my own hospital and on a consultative and referral basis from pathologists who might not be as familiar with the disease and send me cases to look at for diagnostic purposes.

Q. And over the years, have you also participated in consulting for litigation purposes and received specimens of tissue for that?

A. Yes I have.

Q. And what percentage of the consultation historically is related to plaintiff's work as opposed to defense work from the asbestos litigation?

A. Well, it's interesting. You know, neither Dr. Roggli nor myself who handled the lion's share of the consultative practice in that regard at our hospital really sort of bill ourselves as being experts specifically for plaintiffs or experts for defense counsel. We answer the phone. People send us cases. We try to do the best job that we can.

When I first began cutting my teeth, so to speak, as a fellow under Victor Roggli, the vast majority of our cases were from plaintiffs. I can't think of in my early going how many cases, if any, we looked at for defense.

And in the late '80s -- excuse me, the late '90s, most of the contention was based on the diagnosis. Defense counsel did not want to believe that the plaintiff, in fact, had mesothelioma. And in those days, the reports that we wrote -- and we wrote reports for all the, what I call the major players in the asbestos litigation field. We wrote reports for plaintiffs for Peter Angelos. We wrote them for Waters and Kraus. We wrote them for Baron and Budd. We wrote them for Evan Yegelwel and some other Florida based attorneys.

Almost all the work I did early on was for plaintiffs explaining to them that we were able to issue a secure diagnosis of mesothelioma for their clients and, if possible, to go the extra mile and prove that it was related to asbestos.

Q. And how could you as a pathologist prove it was related to asbestos from pathology?

A. There are histologic cues that you can look at. And as pathologists, you don't work in a vacuum. You don't just look at the tissue itself. You examine the entirety of the medical record just as you would if you were dealing with your own patient. And that would include looking at radiographs. That would include looking at occupational exposure history. And then that would also look for things such as plaque or asbestos bodies or increased asbestos fiber burden analysis.

Q. Now, you mentioned something that we're going to talk a

lot about, asbestos fiber burden analysis. Please explain to the court what that is, sir.

A. Okay. Well, asbestos, for those of us who live in industrialized society, we all have asbestos in our lungs. It's everywhere. And with each breath we take just about, unless you live in the pristine mountain environments or out in the desert, chances are you are going to be breathing in everyday asbestos fibers and exhale asbestos fibers. We all have them inside of our lungs.

And we can measure how much asbestos we have by taking lung tissue that has been removed from the body, either in the course of an autopsy or following a surgical procedure, take that to our laboratory, digesting away all the tissue. And what we're left with is the residue in the lungs, the dust and the debris that all of our lungs filter out. And then we're able to take that into our electron microscopy lab and at very high magnification examine those fibers. We're able to bombard them with x-rays and tell not only if there is asbestos and how much, but what type of asbestos.

Q. I see. As a result of all of the consultation, all of the direct patient care samples you have over the years, have you developed in your lab quite a database of lung burden analysis?

A. Yes, we have.

Q. And have you published that and shared it in the peer

reviewed literature, information about that?
A. Yes, we have.
Q. And have you participated in those publications?
A. Yes.
Q. In addition, there's a book on asbestos disease called The Pathology of Asbestos-Associated Diseases, and it's second edition. What's your role on this book, sir?
A. Well, I'm one of the editors, as you can see on the front piece there. And in addition to sort of reviewing what the contributors had for clarity and accuracy, I wrote two chapters in there myself -- no three chapters.
Q. Okay.
A. I wrote the chapters on the site of pathology of mesothelioma and asbestos diseases. I wrote the chapter on mesothelioma itself, and on asbestosis.
Q. Now, this book is in its second edition. Is there another edition coming out soon?
A. Yes, sir. We have a third edition with Springer Verlag. All the chapters are in receipt with the editorial board over in Germany, and we're trying to get this out as soon as possible.
Q. There's also a book called Malignant Mesothelioma of which Andrea Tannapfel...
A. Tannapfel. She's a -- yeah, this is a book that was published by the leadership of the German mesothelioma

registry and reviews in cancer research.
Q. And did you write a chapter in this book, sir?
A. I did.
Q. On what subject?
A. On the mineralogy of asbestos.
MR. SCHACHTER: At this point, Your Honor, we tender this witness as an expert in pathology, asbestos disease, and asbestos fiber type mineralogy.
MR. FINCH: No objection, Your Honor.
THE COURT: Thank you. He will be admitted as such.
Q. The areas I would like to elicit your opinions are three -- on are three.
First, I'd like to get a very clear scientific understanding of the mineralogical difference between the various fiber types. Will you be able to provide us some information on that?
A. I'll certainly try.
Q. And have you prepared some slides that might help illustrate that?
A. Yes, sir.
Q. Secondly, I'd like to call upon your experience in looking at so many lungs or the lung tissue from people who have been in litigation to tell us what we're likely to find in litigation cases. And will you be able to help us with that?

A. I'll certainly try.
Q. And the last thing I'd like to elicit your opinions on is whether exposure to chrysotile end products and specifically gaskets and packing contribute to cause mesothelioma in humans. That's sort of a subject you've written on; is that correct?
A. I don't think I've published with that in the title, but that is something that our group has looked at.
Q. Okay. Do you have opinions on that --
A. Yes.
Q. -- then based upon your research?
A. Yes.
Q. Okay. We'll get into that.
Let's start with the mineralogy of asbestos. How do we divide up the mineral families of asbestos, sir?
A. Well, first off asbestos is not a mineralogic term per se.
Q. Okay.
A. I mean, the name for minerals is usually based on -- either on sort of an honorific name for the individual that discovered it or the properties that the mineral might have or owing to the area where the fiber was found.
But asbestos more properly is a -- it's a commercial and regulatory term. It's not something that -- people use it interchangeably.

Q. Uh-huh.
A. But asbestos is more of a regulatory and classification term for a group of naturally occurring minerals.
Q. All right. So --
A. And these are --
Q. What are the two families?
A. These are -- these are regulatory terms for a group of very useful mineral species that have been used to antiquity. You know, anywhere from the aboriginals in Scandanavia to the pharaohs to the Roman times. They've been used for thousands of years. And now as our mineralogists have gotten more familiar with them, they've formed a classification system for the actual mineral species that we now recognize are regulated and classified as asbestos.
Q. Let me ask you this. Is it important for understanding causation issues to be specific about the mineral species we're dealing with?
A. It is. Because not all asbestos is -- this sounds sort of silly to say. But not all asbestos -- they're not a group of substances that have identical physical properties, mineralogic and bio -- or physical chemical characteristics or chemical composition.
Q. What are the two families, sir, so we can move on?
A. Okay. I'm sorry. In broadest view, the species that are regulated and classified as asbestos are a group -- there's

the serpentine group, meaning that they have a curly, snake-like appearance, and the amphibole species.

Q. And the ones that are used commercially purposely to make products, of all the various forms, the commercial forms are what, sir?

A. In modern times the commercial forms are amosite and crocidolite. And those are fibrous forms of naturally occurring fibrous silicates

Q. And those are the amphibole commercial fiber types?

A. Yes, sir.

Q. And the serpentine commercial fiber type is what, sir?

A. Is chrysotile.

Q. Have you brought us some pictures of what these look like in the macroscopic form? They're rocks, right?

A. Yes, they're -- yeah, they're minerals. They're naturally occurring fibrous silicates.

Q. Okay.

A. And chrysotile or yellow -- white asbestos is that which we see on the left-hand side of our field there. Riebeckite is the name of the nonfibrous form of what we recognize when it's in its fibrous habit as crocidolite.

And amosite is the fibrous form of the mineral cummingtonite grunerite. And amosite itself is also not really a mineralogic term. Amosite is an acronym, Your Honor. The A-M-O-S stands for the asbestos mines of South Africa. So

it's a species that's only found in South Africa.
Q. You brought us some microscopic pictures of the various fiber types.
A. I have.
Q. What is this, sir?
A. This is a photomicrograph taken on a scanning electron microscope of chrysotile fibers. Again, we mentioned these are classified as serpentine, and they have a curly-Q worm or snake-like appearance.
Q. And just so we understand the scale, what is 15um that's up there. What does that mean?
A. That's actually, the letter right after the 50 is not a U. That is a mu (phonetic). So that's 50 microns. And a micron is a millionth of a meter or ten thousandth of a millimeter. So again very, very -- these are taken at extremely high magnifications. These are very, very small fibers.
Q. Are the individual asbestos fibers visible to the naked eye?
A. No, sir.
Q. You have a picture microscopically of what amosite looks like.
A. Yes, sir. And by contrast, the amphiboles have a much more rigid, brittle, and needle-like appearance. And that's what we're seeing here with -- at our digest showing amosite

as opposed to the curly, pliable --
Q. Okay. I'm sorry to flip that.
And this one is crocidolite. Which family is it in?
A. Crocidolite resides in the same family as amosites. It's an amphibole.
Q. And then the other -- one other amphibole that we're going to talk about a little is tremolite. Is this the microscopic picture of tremolite?
A. It is.
Q. And is it more similar to the chrysotile or is it more similar to the amphibole?
A. No, you can see right here that this is much more akin in terms of its growth habit to the other amphiboles, being needle-like and rod shaped rather than -- rather than -- rather than curly.
Q. In terms of the crystal structure, is there a difference between chrysotile on one hand and the amphiboles, amosite crocidolite, and tremolite on the other?
A. Very much so.
Q. And can we demonstrate -- do you have a slide that demonstrates that?
A. Yes, sir. May I use the...
Q. Yes, and if it will help to approach there so you don't have to send the laser over people's heads, please come down.
A. I think I can maybe do this. I won't obstruct anybody's

view.

This is the crystal structure of amphiboles. And you can see here in the dark gray here and in the light gray here, there are two bundles of the asbestos fibers. And they have -- and the asbestos fibers amphiboles are composed of the silica tetrahedra, and they have on their inside a layer of cations. And cations are positively charged groups of atoms. And the cations are typically magnesium, calcium, iron. And these are on the inside.

And the cations, they are on the inside. Their location away from the surface confers upon them the ability to be resistant to biodegradation. So when eventually these might get broken down, then you see a -- can see a splitting of the bundles. And then once the bundles are released, they can break down into individual fibers.

Q. We're going to hear about a term called biopersistence. Perhaps it would be helpful if you define that for us now, sir.

A. Biopersistence is the -- it's the amount of time that an inhaled particulate can persist in the body. And that is dependent on a variety of different things: On the size; how it gets into the body; where it gets into the body. And then what it's biochemical or physical chemical characteristics are will also impact on its biopersistence.

Q. For amphibole fibers that make it to the lung, what is

the biopersistence? How do we measure that?
A. The biopersistence of amphiboles is quite long. And these often will have a residence time in the body of many years. They are extremely resistant to biochemical degradation. They're extremely resistant to acid environments. That's what makes them such good compounds for use in industrial applications is they are resistant to chemical degradation. Our bodies break these fibers down biochemically in the acid environment of scavenger cells. And the amphiboles owing to their physical chemical composition are much more biopersistent.
Q. Biopersistence is often measured in half life. I guess that means how long it takes half of them to disappear from the body?
A. Yeah, half life is a term that is used in a lot of different physical chemical settings. It's the amount of time that a substance or a compound will be around. And then the half life is how much time will it take until half of it goes away and then another half goes away until ultimately you're left with nothing.
Q. And is the half life of amphiboles measured in days, weeks, months, decades? What?
A. I think the biopersistence in half life of the amphibole species is measured in months to years.
Q. Now, the chrysotile, how does it differ and what is its

structure?
A. This is -- is what a chrysotile fiber -- cartoon of what a chrysotile fiber would look like. Instead of those parallel bundles, chrysotile is sort of rolled up in sheets like a jelly roll or a scroll. And rather than having the cations on the outside and the magnesium hydroxide groups, they are -- those would be on the inside and resistant to degradation with the amphiboles.

Here we see them on the outside. And let's assume this was a chrysotile fiber that found itself in the cytoplasm of a macrophage. These magnesium hydroxide groups would be readily leached away by the acid environment of the macrophage allowing for the silica sheet to undergo degradation. And it would split -- it would either become amorphous or it would split into small fibrils which then would be swept away very readily by the body.

The upshot of this is it has a much longer residence time in our lungs on the orders of days to weeks.
Q. Okay. Much longer or much shorter time for chrysotile?
A. Yes, sir. The order -- the biopersistence of chrysotile is much, much shorter. In fact, there are some species of short fiber chrysotile like Calidria asbestos which is mined out in California has the most rapid clearance times of a great many different particulates.
Q. All right. Chemically if we were to chart out what the

chemical formulas of these various fibers are, have you brought us a chart that will show that?
A. Yes, sir.
Q. And how does this chart work? Can you explain it to us, sir?
A. Well, the stylized chemical formula for the amphibole asbestos are the silica tetrahedra that are complexed with different cations and hydroxyl groups.
Q. So those are the two?
A. Yes.
Q. Okay. And what is the 22?
A. That's the concentration of oxygen -- of the silicon oxide groups here forming the central part of the core.
Q. So we charted out how many atoms of each of these different chemicals. That will be 7 of iron magneseum?
A. Yes.
Q. Is that what the MG stands for?
A. Yes.
Q. Okay. And 8 of silica and 22 of oxygen and the 2 OHs, right?
A. Yes.
Q. And then crocidolite has a -- is it similar or dissimilar?
A. Crocidolite is quite similar to amosite. The main difference between that you have this sodium. The chemical

abbreviation for sodium is NA. So crocidolite has a sodium peak on it as well.

Q. All right. And tremolite, is it similar or dissimilar to the other amphiboles?

A. Yes. You can see that the -- that the central magneseum of silicate core is present. Tremolite, however, the cation that is complexed with it is calcium.

Q. Now, does this represent in the same manner the chemical formula for chrysotile?

A. It does.

Q. And what is different?

A. Well, the predominant difference is the -- is the absence of iron.

Q. Okay. And so in summary, would it be fair to say that the amphiboles have a different chemical structural and biological -- biologically relevant composition than chrysotile?

A. Yes, sir. They have different crystalline structures They have different chemical composition and different physical chemical characteristics.

Q. Is there a biological consequence of the difference between the amphiboles and chrysotile for mesothelioma analysis?

A. Yes, there is. And I think one of the major factors that influence how injurious an inhaled particle is, in addition to

its size and shape, is its biopersistence.
Q. Okay.
A. And the -- as we just discussed, the biopersistence amphiboles is well in excess of the biopersistence of chrysotile. Therefore, the amphiboles are more pathogenic in terms of resulting in fibrosing disease in the lung as well as malignancies, neoplasia.
Q. Sir, is there any question that the amphiboles, the amosite, crocidolite, and tremolite, are causes for mesothelioma in human beings?
A. Sir?
Q. Is there any question about whether amosite, crocidolite, and tremolite is causes for mesothelioma in human beings?
A. Is there a question?
Q. Yeah. Do they cause it?
A. Yes. Yes, they do.
Q. Okay. When we discuss chrysotile, I want to be very specific. Is there a distinction we should make between chrysotile ore and chrysotile fibers themselves if we want to be scientifically rigorous in our discussion?
A. I would prefer that there would be. I think it's important that as we have our discussion here, we discuss differences between chrysotile fibers as you might encounter in a -- in a laboratory situation. I think there is a difference between chrysotile and commercial products. And I

think there is a very real need to discuss the potential for disease induction by chrysotile mine dust. I think those are three separate entities and all have different abilities to induce disease.

Q. Okay. In the context of mining populations, has the chrysotile dust or the ores, or whatever is in it, in the mining populations been associated with an elevated rate of mesothelioma?

A. Yes.

Q. And is that attributable -- what's that attributable to?

A. The current feelings amongst, I think, the people that have most closely evaluated this is that there's no data to suggest that exposure to pure chrysotile dust without its -- without any type of contaminating species does not cause mesothelioma. And that the remainder of the chrysotile-associated mesotheliomas and asbestosis are related in the presence of contaminating amphiboles.

Q. Okay. In the mines we've heard about fiber years as a measure of how much exposure. What kinds of -- go ahead.

A. And I think I need -- I think I need to back up on that.

Q. Please do, sir.

A. Because chrysotile is a mineral that is mined worldwide, I think in every -- every country but -- or every continent but Antarctica. Chrysotile is mined in South America. Chrysotile is mined in the United States of America. It's

mined in Canada. It's mined in South America -- or South Africa. It's mined in Southeast Asia.
Q. All right. And do all those mining populations have an increased incidence of mesothelioma?
A. No, they don't. And not all those mines suffer from contamination of the chrysotile ore with contaminating amphibolic species.
Q. All right. Is there an area where there is a contaminating amphibolic species?
A. Yes.
Q. Where is that?
A. That is principally in the chrysotile mining areas of the province of Quebec in Canada.
Q. And are there some mines that do have the contamination and others that don't?
A. Yeah. It seems to be a very geographically -- the preponderance or degree of contamination with noncommercial amphibole tremolite seems to be a function not only of the different mine locations, but within the locations of those particular mines within that area.
Q. Okay.
A. And some of the chrysotile mines in Quebec do not appear to have substantial tremolite contamination at all.
Q. All right. At what level of exposure do you need to have to -- in the mining context to the tremolite contaminated

chrysotile to be at risk of increased mesothelioma?
A. As we discussed earlier, the biopersistence of chrysotile is less than the amphiboles, and that confers upon it a much lower degree of pathogenicity. Therefore, it doesn't take much asbestos exposure to result in the induction of diseases like mesothelioma if the asbestos that you've been exposed to is an amphibole like amosite or crocidolite. That's not true with chrysotile. You would need to have hundreds of fiber years of exposure as would typically be sustained in the induction of fibrosis in the lung or asbestosis. And again, we're talking in the order of hundreds of fiber years.
Q. All right. Of course, the issue in this case, I don't believe there are any miners that have been claimants. We're talking about an end product. Does the level of exposure for end products like gaskets, packing, come anywhere near to the levels where an increased risk from any kind of chrysotile exposure has been demonstrated?
A. I don't believe so.
Q. Another issue that's arisen in this case is the issue of idiopathic mesothelioma. Does mesothelioma occur without asbestos exposure?
A. It can. And idiopathic is the term we use in medicine where we don't know what causes a particular disease. They just sort of arise sporadically within our society. Not all -- we think of lung cancers as being overwhelmingly

associated with cigarette smoking, yet there are a handful of people who don't have radon exposure, don't have any other means for getting lung cancer. We look upon those as idiopathic. An analogous situation is within the mesotheliomas in that there are mesotheliomas that -- where we can't identify an exposure to asbestos. We can't demonstrate asbestos in their lung tissue. And we can't come up with another plausible cause for them to develop mesothelioma. There are a handful of people whose mesotheliomas are neither related to asbestos exposure nor idiopathic and those are rare. Those are people who have undergone chest wall radiation for soft tissue sarcomas. They are people who have had chronic pleural effusions or fluid in the chest from tuberculosis. But again, those are rare. But yes, there is an established or ballpark figure of a rate of idiopathic mesothelioma in the United States.

Q. And what is that rate just generally, sir?

A. Well, it has -- you have to sort of break that down both in terms of topography where the mesotheliomas occur and the gender. In men pleural mesotheliomas are at least 80 percent related to an exposure to asbestos.

Q. Okay.

A. In women it's much less. Peritoneal mesothelioma is mesotheliomas that arise in the serosal membrane that invest the wall of our abdomens and the visceral that are contained

in our abdominal cavity. Perhaps only 25 percent of mesothelioma -- of peritoneal mesotheliomas in women are caused by asbestos. And maybe three times that, maybe 50 to 60 percent in men.

So the association for asbestos-related mesotheliomas is greatest in men in the pleura followed by greatest in men in the peritoneum, and then with women bringing up the rear at both sites.

Q. Thank you. I'd like to turn to the area of what your studies have shown, your fiber burden studies have shown about the nature of the asbestos found in the lungs of people, okay.

A. Yes, sir.

Q. And have you brought us information -- you've published on this topic, right?

A. I was -- I was a co-author and my work went into this, yes.

Q. And this is an illustration of a table that appears in your article. What's the nature of that article or the name of it? Just identify it if you can.

A. It was a case where we looked at 1,445 cases of mesothelioma where we had an occupation or a reliable occupation for the individual as well as a fiber burden analysis.

Q. And were a lot of these litigation cases?

A. They were all litigation cases.

Q. All litigation cases, and primarily from defendants or plaintiffs or what?
A. Both.
Q. Both. All right. And did you break down what you were finding in the lungs by occupation?
A. Yes.
Q. And what were the occupation or profession categories that you chose?
A. Well, you can see that on the bottom axis. These were commercial insulators, pipefitters, boilermakers, ship -- people who worked in shipyards, electricians, construction workers, maintenance workers at various different types of factories and other concerns, as well as individuals who had served in the United States navy, as well as automotive workers, household contacts, and then just people who had -- who really didn't have an occupation that we could pinpoint.
Q. Okay. On this chart there is red, something that's in red. What does the red illustrate?
A. The red illustrates what percentage of cases had increased commercial amphiboles.
Q. Would it help if you came down here to see it or can you see fine from there?
A. I think I'm okay.
Q. Okay. Great. I just want you to be comfortable. And when you say increased levels, elevated levels, what do you

mean by elevated levels?
A. Well, as I mentioned, we all have asbestos in our lungs assuming that we have grown up in an industrialized society. And Dr. Roggli and his mentor Dr. Pratt, and others were amongst the first, at least for our lab, to determine what a background level of asbestos was. And that is somewhere between zero and 20 asbestos bodies per gram of wet lung tissue. We can -- when we measured the amount of -- uncoated fibers, too, in addition to asbestos fibers.
A. Okay.
Q. We then compared what we saw amongst insulated -- the insulator cohort and the pipefitter cohort and contrasted those to our control population.
Q. Okay. So in what percentage of insulators did you find an elevated level of commercial amphiboles in the lungs of the people who had mesothelioma?
A. A hundred percent.
Q. For pipefitters, approximately what percent did you find had an elevated level of amphiboles in their lungs?
A. As you can see, they are in excess of 90 percent.
Q. And this chart reads down similarly. Did you consistently find amosite in the lungs of the people that were occupationally exposed to asbestos?
A. Yes, we did.
Q. And did you draw any conclusions from that?

A. Well, the conclusion was that that's what caused their mesothelioma.
Q. All right. Now, you also looked for other fiber types, correct?
A. We did.
Q. Noncommercial amphiboles, that would be tremolite and related noncommercials.
A. Yes.
Q. Did you ever get a consistent pattern of above background levels of the noncommercial amphiboles in the majority of any occupational category?
A. No. You can see that in -- depending on the population, as high as 60 percent of electricians may have had elevation of noncommercial amphiboles; whereas, automotive workers or maintenance workers, there was a minority of those individuals who had an increase in the noncommercial amphibole species.
Q. And did you also look for chrysotile?
A. We did.
Q. And did you find a consistent pattern of above background levels of chrysotile in any of the occupational categories?
A. No. Again, you can see that there was considerable variance amongst the occupational groups there.
Q. Now, sir, your research that you've talked to us about is focused on the lung. Is the lung the place where mesothelioma occurs?

A. No, sir.
Q. Then why is -- where does mesothelioma happen?
A. Mesothelioma typically arises in the pleura. The pleura is the membrane that lines the chest cavity. And that -- that's called the parietal pleura and that's typically where mesotheliomas originate.

There is also the visceral pleura which is the membrane that lines the lung. But most of the mesotheliomas arise in the parietal pleura. but --
Q. Well, then, why -- go ahead.
A. But the portal of entry for the asbestos fibers is through inhalation; and these fibers, assuming that they are of the right size and confirmation, get deposited at branch points in the parts of the lung that exchange gas and then they make their way out either mechanically or through macrophages, macrophages being a specialized scavenger cell, and these move all around the body.
Q. Well, why aren't you looking for the fiber burden in the pleura?
A. Well, I think there have been studies that have shown that levels of amphiboles in the pleura are predictive by levels of amphiboles in the lung.

The other reason is we don't have control values for what normal ranges of amphiboles or nonamphibole asbestos is in the pleura.

Q. All right. And why is it important that you have a control value of what the background levels are in the pleura in order to use pleural tissue?
A. Well, if you don't know what the levels are in a control population or a population that doesn't have disease, how are you -- how are you going to make any sense out of a level of asbestos in someone with disease?
Q. There is -- there's some studies published by Suzuki and others that note that short chrysotile fibers are found in the pleura. Are those informative or -- in proving that there is causation by chrysotile of mesothelioma?
A. Not at all.
Q. Why not?
A. Well, I think that if you -- first off, short fiber chrysotile is believed by the vast majority, I believe, of scientists who work in this area as to be nonpathogenic. And if you were to believe the Suzuki model where lots of short fiber chrysotile is the fiber responsible for mesothelioma -- in the Suzuki model chrysotile would be the major cause of asbestos, and I don't believe that that's true.
Q. The major cause of mesothelioma?
A. Yes, major cause of mesothelioma is amphibole asbestos, not --
Q. And have you and your group published the view that amosite exposure is the number one cause of mesothelioma --

A. Yes.
Q. -- in the United States?
Huh?
A. I don't know -- I mean, I think that is implicit in several of our writings.
Q. Thank you. I have a picture here. What is that, sir?
A. Okay. This might take a little bit of orientation purposes. This is a piece of -- and this is not a photomicrograph.
This is a piece of the chest wall that has been removed from somebody. We are looking at it with the naked eye. This is so -- this is the inner lining of the chest wall. You can sort of make out where the ribs are running. There is a rib running there. And this here is the parietal pleura. And what we are seeing here, these black areas here are called black spots.
Q. Yes, sir.
A. And these black spots are areas where there are stomas or openings in the pleura. And what is concentrating there is carbon pigment. We all have carbon pigment. We have dark areas in our lung tissue. We have dark areas in our pleura. And that has to do with our scavenger cells having scavenged black pigment that we inhale, motor vehicle exhaust, industrial soot, and so forth, and it becomes concentrated at those areas.

And Dr. Boutin, a French expert in mesothelioma, has measured the amount of asbestos fibers at these areas and found out that they consist exclusively of amphiboles and that amphiboles are distributed heterogeneously throughout the rest of the pleura.

This article, I think, is one of the major articles that gives credence to why mesotheliomas develop here in the parietal pleura. And as lung -- and as lung values are predictive of pleura values, that is why we use lung tissue rather than pleura.

Q. Now, one of the criticisms that I think is in the briefing on your studies is that chrysotile, because it clears much more rapidly, isn't found in fiber burden studies. So that if it were causing it, your fiber burden studies would be irrelevant. How do you respond to that criticism?

A. I've heard that before and we tend to view that as, quote, the hit and run. The hypothesis is that chrysotile, being a form of asbestos potentially able to cause mesothelioma, goes into the body, does its work on disrupting cell DNA. It's broken down by the body and then vanishes. I don't think there's any scientific proof of that. And a substance that doesn't reside very long in the body in my opinion isn't likely to cause genetic damage, at least in a real-time example within a patient.

Q. Now, from the studies that have now existed, do we

actually know the complete biomedical causation string that leads from asbestos to the development of the mesothelioma?
A. No. And I think you bring up a -- one of -- an area in biomedical science not just from mesothelioma, but for, in fact, all of -- all of cancer biology. It's the steps that are undertaken that result in a cell becoming transformed to where a patient gets the clinical entity of having cancer, that's not worked out, I mean completely worked out. There is an explosion of knowledge and a lot of what are believed to be individual cancers we now recognize are actually a family of cancers with different mutations, different gene rearrangements. Very -- very complex at the -- at the molecular level.
Q. Do we know at the molecular level how many changes are necessary in order to create a mesothelioma?
A. I think for solid tumors across the board, somewhere between 26 and 30.
Q. And do we yet know which precise genetic changes have to occur and in what order before you get this very rare disease mesothelioma?
A. Maybe somebody like at Brooke Mossman's lab at the University of Vermont, they might be as close to anybody to knowing what all the sequences are. I don't.
Q. Does the fact that chrysotile in laboratory studies can cause damage to a cell mean that it necessarily causes the

right kinds of damage that can create all of the genetic mutations necessary to cause mesothelioma?

A. I don't believe so. And the reason for that is not all experimental models of -- that result in the induction of carcinogenesis have real world in vivo or in patient applicability.

Our bodies undergo premalignant genetic changes every day from cosmic radiation, from ultraviolet light, from the substances that we eat, from the viruses that we are infected with. So we undergo genetic damage every day. Our bodies develop cancer cells or cells that are on their way to becoming cancer cells every day. And fortunately, we were blessed with mechanisms to repair our DNA where the genetic damage occurs, and our immune system is also competent at clearing those cells.

So even though we might generate a cancer cell, the -- those don't necessarily result in that particular individual getting the clinical syndrome of cancer.

Q. One of the themes that we've seen in the briefing from our learned adversaries here is that a number of public health agencies have issued statements about whether chrysotile may be a cause of mesothelioma. From a scientific standpoint, how do you assess those in making causation decisions in an actual case of whether it is a cause?

A. I think that scientists such as myself who work in a

hospital and are -- and the research that we do, we are perhaps in the same church but in a different pew.

The organization such as -- and I assume you're talking about IARC, the International Agency for the Research on Cancer and other public health agencies.

Q. Sure. Right.

A. I think that public health agencies out of an abundance of caution prefer to take everything back to a very fundamental level and say if it could possibly cause cancer, if it's been observed to cause cancer, it is best for the public not to be exposed to that type of material and it's best to be removed from the workplace. And that's not an entirely unreasonable opinion to take if you're someone who is charged with the safeguarding of the public health.

I think it's different, though, when you are as a scientist looking at the disease in an individual case or in a group of cases.

Q. Sir, another discrete issue that's come up is that one of the IARC monographs relies heavily on a recent article by Loomis and Dement that has some information about the Marshville plant, and I won't belabor this because we went through it a little bit with another witness. But do you have any information to shed on the Marshville plant from your perspective of fiber burden analysis?

A. My take on reviewing the Loomis and Dement article about

Marshville is that this was a report alleging mesotheliomas to have developed in a cohort of individuals exposed only to chrysotile. And there are only a handful of -- if you look through the literature, there are only a handful of cohorts that are alleged to be exposed solely to chrysotile, and this Marshville plant was one of them.

And when you look at it, though, the Marshville plant wasn't a plant where they used only chrysotile. They may have used predominantly chrysotile. But if you go back and you look at the documentation that is available for that plant that was supplied by its owners, there was both amosite and crocidolite used there. And my colleague, Dr. Roggli, actually analyzed the lung tissue of one of the women from the Marshville plant.

Q. What did he find?

A. He found amosite. And so I think that -- I think the fact that chrysotile -- or crocidolite was used and amosite were used at the plant, and I think the fact that amosite was actually demonstrated within the lung tissue of one of the workers there who developed mesothelioma, I think it compromises its ability to stand as an example of a chrysotile-only plant.

Q. Sir, you told us that in mine -- some mining populations there is in excess -- chrysotile mining populations, there's been an elevated risk of mesothelioma. Why doesn't that mean

that there must be a danger of mesothelioma from gaskets and packing?
A. Those are two different sets of occupations with -- an industrial hygienist will bear this out -- with completely different exposure levels to the mineral dust. Miners and millers of the mineral dust are exposed to hundreds of fiber cc years. And this was looked at in -- by the workers in Quebec where the women in the mining areas, the women developing mesothelioma, regardless of whether they worked in the factory or they just were residents of the area or had exposure levels measured in the hundreds of fiber CC years.
Q. Sir, as a bottom line question, as an expert in asbestos medicine, are you aware of any scientific -- scientifically reliable methodology that would allow the formulation of a reliable opinion that the exposure from gaskets and packing is capable of causing mesothelioma in human beings?
A. I am not.
MR. SCHACHTER: Thank you, sir.
MR. FINCH: Your Honor, just give us a second to switch.
THE COURT: Yes.

CROSS EXAMINATION

BY MR. FINCH:
Q. Good afternoon. Nate Finch for the asbestos claimants committee.

Good afternoon, Dr. Sporn.
A. Good afternoon, Mr. Finch.
Q. You have testified in 45 -- 40 to 50 cases in asbestos litigation, correct?
A. Deposition and courtroom testimony. About, yes, sir.
Q. Yes. And you talked on direct about how you had done work with your colleague, Dr. Roggli. Isn't it true that in terms of the time you've spent in testifying, 80 percent of that has been for a defendant in asbestos cases, correct?
A. Yes.
Q. You have testified at the request of Garlock, obviously, here today, correct?
A. Yes.
Q. You've testified at the request of Ford which made chrysotile-containing brakes, correct?
A. Yes.
Q. Honeywell, correct?
A. Yes.
Q. Pneumo Abex?
A. Yes.
Q. General Motors?
A. Yes.
Q. Chrysler?
A. Yes.
Q. All those companies make chrysotile-containing brakes,

correct?
A. Yes.
Q. And Warren Pumps was a company that used asbestos-containing gaskets and packing on or around its equipment. You testified for them, too, correct?
A. My testimony for Warren Pumps, I believe, was not so much in their defense but in insurance litigation. I wasn't an expert, I don't believe, for Warren Pumps.
Q. You were an expert for Warren Pumps in insurance litigation not in a lawsuit where a plaintiff was suing Warren Pumps.
A. Yes.
Q. You're not an epidemiologist, sir, are you?
A. I'm not.
Q. You have not conducted any epidemiological -- analytical epidemiological studies of asbestos and disease, correct?
A. I have not.
Q. Of your publications on asbestos, none of them specifically deal with asbestos disease and the causation of asbestos disease, correct?
A. Correct.
Q. You might want to speak up a little bit.
A. I'm sorry.
Q. You would agree with me that 95 percent of the asbestos ever used in products in the United States was chrysotile

asbestos.
A. Yes.
Q. You are suspicious as to whether there is any such thing as pure chrysotile outside of a laboratory setting, correct?
A. No, I don't know if I'm suspicious. I think that -- I think there -- there is ample evidence there is pure chrysotile. I mean, if I may, I think you mean pure chrysotile in the absence of its natural contaminant tremolite.
Q. Do you recall being deposed in this case, sir?
A. I do.
MR. FINCH: Would you flip up the deposition, please, John. Page 39.
Q. "So you don't have an opinion about whether pure chrysotile, hypothetically, can cause plaques or asbestos?
I think that -- I'm suspicious, outside of the -- of the laboratory setting, whether there really does exist such an entity of pure chrysotile."
Was that the question you were asked and the answer you gave under oath in your deposition?
A. It was.
MR. FINCH: Take that down.
Q. You've also testified that some tremolite is unavoidably retained in the milled final product, correct?
A. Yes.

Q. You would agree that asbestosis is a disease caused by asbestos exposure, correct?
A. Yes.
Q. And that chrysotile fibers in suspicious doses have been shown to cause asbestosis in humans, correct?
A. I think we have to be clear. There is a difference between chrysotile fibers and chrysotile mine dust. Chrysotile mine dust has been shown to cause asbestosis.
Q. You would agree with me that chrysotile fibers can cause lung cancer.
A. Again, I think chrysotile -- again, we have to be very specific here. Is that chrysotile fibers in animal models? Yes. But if we're talking about humans, I think we have to be very careful to get our terms precise and talk about chrysotile mine dust.
Q. So it's your testimony that chrysotile from a finished product cannot contribute to asbestosis in a human being; that's your testimony?
A. Yes.
Q. Okay. You would agree with me that chrysotile fibers do get to the pleura.
A. Some fibers do, yes.
Q. And you agree that chrysotile fibers have been shown that they can damage chromosomes, correct?
A. Again, I think that once -- yeah, I think that all -- all

of these -- the first two, again, I would couch that in saying is that chrysotile fibers from chrysotile mine dust -- in mine dust cause asbestosis and lung cancer. Chrysotile fibers can get to the pleura and chrysotile fibers in laboratories can damage chromosomes.

Q. And they can also interfere with the body's cancer defense mechanisms, correct?

A. Probably, yes.

Q. You talked about biopersistence. I believe that it's your testimony -- or it's your view -- it's certainly Dr. Roggli's view -- that ionizing radiation can cause mesothelioma.

A. Yes.

Q. Ionizing radiation doesn't persist in the body, does it?

A. No, it doesn't.

Q. And isn't it true that cigarette smoke doesn't persist in the body yet cigarettes cause lung cancer, correct?

A. But I think that those aren't necessarily comparable in terms of what they -- how they damage or how they cause their damage.

Ionizing radiation, I think, can cause much more severe damage to the DNA of cells. I mean, ionizing radiation is used to kill off the cancers. I think it causes -- and I think that it's not analogous just because ionizing radiation can cause genetic damage.

Chrysotile, which can cause genetic damage, are miscreants on the same level. Certainly not everybody who has -- receives ionizing radiation gets a second malignancy.

Q. Let's talk a little bit about tremolite and chrysotile in finished products. You don't go out and test products to see whether they have tremolite contamination in it, correct? That's not part of what your expertise is.

A. No.

Q. And you never tested Garlock gaskets to see to what extent they have tremolite in them, did you?

A. I didn't.

Q. You agree chrysotile causes mesothelioma. You testified to that before without qualification, correct?

A. I think I have testified -- if I have said that, it's -- my opinion is that chrysotile mine dust causes mesothelioma.

Q. Do you recall testifying in a trial in the city of Baltimore a few years ago in a case called *Fitzgerald versus AC&S*?

A. I don't. Oh, yeah, yeah. Yeah, I do.

Q. And you were asked this question: "But you do agree that chrysotile causes mesothelioma.

"Answer: I do agree that chrysotile causes mesothelioma."

That was the question; that was your answer.

A. That was my answer.

Q. There was no qualification in that answer, correct?
A. No, there was not.
Q. You also agree that there is no safe level of exposure to chrysotile fibers longer than 5 microns, correct?
A. Well, there -- a safe level of exposure to chrysotile fibers longer than 5 microns has not been established. I don't believe -- that's not the same as saying that it does not exist.
Q. Same trial. "And you agree there is no safe level of exposure to chrysotile fibers longer than 5 microns, correct?
"I do.
"I am sorry?
"Yes, sir."
That was the question that you were asked; that was your answer in front of a jury in Baltimore three and a half years ago, correct?
A. Correct.
Q. You would agree that a threshold level of exposure to asbestos below which mesothelioma will not occur has not been determined by science, correct?
A. It has not. But again, that does not mean that one does not exist especially for chrysotile.
Q. You define short fiber chrysotile as fibers shorter than 5 microns in length, correct?
A. Correct.

Q. You have testified before that a person who has mesothelioma, all of their exposures to asbestos other than to short fiber chrysotile would contribute to the development of the disease, correct?
A. Correct.
Q. You agree that any exposure to long fiber chrysotile would pose some risk of mesothelioma.
A. Again, I don't know if I was given the opportunity to flesh out my answer; but, yeah, if that's what you are putting up from my deposition, then I must have said it.
Q. You would agree with me that all of these scientific and research agencies have concluded that chrysotile causes mesothelioma. They've all stated that in publicly available pronouncements. Whether it's an IARC monograph or the World Health Organization or the United States Surgeon General, all of those agencies on that chart have said that chrysotile asbestos causes mesothelioma in human beings.
A. Yes, they have.
Q. Okay. Let's go -- you talked a little bit about public health agencies and regulatory purposes. You would agree with me that the International Agency for Research on Cancer doesn't regulate cancer. It tries to establish what is a confirmed human carcinogen, correct?
A. That's what they say.
Q. They don't pass laws or regulations or rules to try to

reduce carcinogens or the epidemic of cancer in the world. They look at what causes cancer, right?

A. They look at what they think causes cancer or what they have written causes cancer.

Q. Okay. The National Cancer Institute, likewise, doesn't regulate cancer in the United States and it has stated that chrysotile causes mesothelioma, correct?

A. I've not seen that.

Q. You don't know one way or the other, correct?

A. I don't.

Q. All right. Did Garlock ever show you material data safety sheets that it put out for its chrysotile sheet gaskets?

MR. SCHACHTER: Your Honor, we have to object to the introduction of the Garlock MSDS. Subsequent remedial measures are not admissible to prove liability.

MR. FINCH: Your Honor, this is not -- first of all, this is not a liability issue in this case.

Secondly, it's not a subsequent remedial measure. There were plenty of people that were exposed to Garlock gaskets after the date of this document.

It's an admission by Garlock as to what working with its gaskets can do. An MSDS sheet doesn't go to end users. It's a document that's created for -- for -- by Garlock and it doesn't -- it's not like a label on a gasket or it's not like

a label that goes to the end using public. It's sent out by Garlock and it's an admission on the question of causation, but it is -- it is not a subsequent remedial measure.

MR. SCHACHTER: If I may, Your Honor, just briefly.

As we briefed in our *Daubert* briefing, MSDS has been uniformally rejected as proof of causation. These are required documents under government regulations requiring to be protective and over -- erring on the side of over protection just like all other protective measures. And we therefore object to the use of them.

MR. FINCH: Your Honor, at the time this document was written, the government did not require the exact language Garlock put in its MSDS.

THE COURT: We'll admit the document and let you examine about it.

MR. FINCH: Thank you. And there is an exhibit number. I will give you the exhibit number when we offer it in our case, but I just want to show it to Dr. Sporn.

Q. This is a document, chrysotile sheet test MSDS written by Garlock. It talks about chrysotile asbestos in the gaskets. "What are the health hazards? Chronic breathing of amounts of asbestos fibers can cause lung disorders such as asbestosis, pleural plaque, lung cancer, and mesothelioma.

"Dust from sheet should be treated as free asbestos. Secure the area. Clean up using HEPA-filtered vacuum or wet

sweep. Do not clean up in a method that creates dust."

That's what Garlock said in its MSDS. It never showed that to you, Dr. Sporn?

A. No.

Q. You talked a little bit about Calidria asbestos on direct examination. Do you recall that?

A. Yes.

Q. That's made by a company called Union Carbide and the Calidria Corporation, right?

A. Correct.

Q. Has Union Carbide ever showed you its MSDS on Calidria asbestos?

A. No.

Q. "Over exposure to chrysotile asbestos has caused damage to lungs, (asbestosis), lung cancer and mesothelioma of the pleura and peritoneum. Symptoms, which are usually not manifested until 15 to 20 years after exposure, include labored breathing, chest pains, weakness, and chest tightness."

That's what Union Carbide has said about its Calidria asbestos. They never showed that to you?

A. No.

Q. At the end of my direct exam -- my cross exam, I'm going to ask you some questions about the health impacts on people with mesothelioma. So we'll get to that in a little bit. But

I want to talk to you a little bit about your fiber burden studies.

The -- you acknowledged on direct that fiber burden analyses look at what's found in the lung, correct?

A. Correct.

Q. And not what's found in the pleura, correct?

A. Correct.

Q. And that mesothelioma obviously occurs in the pleura, correct?

A. Correct.

Q. All right. Now, your case series of 1,445 cases that you and Dr. Roggli are authors of, that was -- that was published in the peer reviewed literature, correct?

A. Yes.

Q. And while it's not an analytical epidemiology study, it's a case series that you at least rely on to inform your views about the causation of mesothelioma, correct?

A. Correct.

Q. And you would agree with me that by 1960, the world came to the conclusion that there was -- that crocidolite asbestos could cause mesothelioma based on a paper published by Wagner in 1960, right?

A. Correct.

MR. SCHACHTER: Objection, Your Honor. He's going into the history of mesothelioma which is beyond the testimony

of the other witnesses.

THE COURT: We'll let him answer the question if he can. Overruled.

A. Repeat, please.

Q. My question was the world literature, the medical and scientific community in 1960 concluded that crocidolite asbestos could cause mesothelioma based on a seminal paper by Dr. Wagner in South Africa which was published in 1960, correct?

A. Yes.

Q. Is that yes?

A. Yes.

Q. Okay. That was a case series. That wasn't an analytical epidemiology study, correct?

A. Yes.

Q. Okay. Now, on the question of fiber burden analysis, this is an article that you and Dr. Roggli published together with Vollmer and Kelly Butnor in 2002 called "Tremolite and Mesothelioma."

A. Right.

Q. And it discusses the presence of tremolite in people's lungs. And the group of people that you're looking at is a subset of your 1,445 case series; is that correct?

A. Right. Yes.

Q. Okay. And am I correct in your 1,445 patient -- or

litigation case series, you only had lung tissue to do fiber burden analysis in about 280 people.
A. I can't remember the exact number that we had.
Q. It was less than 300 though, right?
A. Yes.
Q. Okay. And of that -- so only about a fifth of them did you have any kind of lung tissue to do a fiber burden analysis on.
A. Right.
Q. And that's pretty common in asbestos litigation. A lot of times you don't have lung tissue because you're only going to get it if the person dies and there's an autopsy or if there is an extrapleural pneumonectomy, right?
A. Correct.
Q. So a lot of times you'll have a patient with mesothelioma who might testify at a trial or might be alive or they die and for whatever reason there wasn't an autopsy so you don't have the lung tissue to do the fiber burden analysis, right?
A. Right.
Q. And of that 280 odd cases where you did have the lung tissue to do the fiber burden analysis, only ten of them were brake workers, right?
A. Yes.
Q. So when you showed the judge the chart with the occupations and what percentage of them had commercial

amphiboles in their lungs and you had the brake workers, you had a heck of a lot more insulators than you had brake workers. You only had ten brake workers to look at, right?
A. Right. And that's because this is not a common disease amongst brake workers. This is a common disease amongst insulators.
Q. Let me -- in your paper, 2002 paper, Roggli, Sporn -- I'm cutting out the middle two authors to make it fit on the screen, but you know you had two co-authors.
A. Butnor and Vollmer.
Q. No disrespect to Butnor and Vollmer, but to make it simple, you recognize that as a statement out of your paper, correct?
A. Yes.
Q. Okay. And what you wrote along with Dr. Roggli is that "although chrysotile concentrations were also strongly correlated with tremolite fiber burden, chrysotile accounted for a smaller percentage of the tremolite deviance. In this regard it should be noted that chrysotile does not accumulate in lung tissue samples to the extent that the amphiboles do, as it is broken down into smaller fibrils that are more readily cleared from the lungs. These smaller fibrils would have been missed by our technique."
And you're talking about your technique that your laboratory at Duke uses, correct?

A. Yes.
Q. "Since we only counted fibers that were greater than 5 microns in length. Furthermore, chrysotile fibers split longitudinally."
That means they split long ways, right?
A. Yes.
Q. Okay. "In vivo."
That means in the living animal, correct?
A. Right.
Q. Okay. "To produce long fibers that are less than 1 micron in diameter."
And so what you mean in there, you can have chrysotile fibers that are longer than 5 microns long, but they're too thin to see by your microscopic techniques because they're less than 1 micron in diameter, correct?
A. Correct.
Q. All right. "Such long, thin fibers would likewise be missed at the screening magnification that we use. Since chrysotile content is poorly detected by SEM," that's scanning electron microscopes, "and fiber burden is a poor indicator of total chrysotile exposure, other information must be sought to address this question.
"The best indicator of chrysotile exposure is an occupational history."
That's what you wrote in 2002, correct?

A. Correct.
Q. You're familiar that other people have written papers on the limitations of drawing etiologic inferences based on measurement of asbestos fibers from lung tissue, correct?
A. I've not seen this particular one. I've heard that.
Q. Okay. So you're not familiar with -- you're familiar with Irving Selikoff, correct?
A. Sure.
Q. He published a book called "The Third Wave of Asbestos Disease" in about 1990 or 199 -- early 1990s. Do you remember that?
A. I've never read it.
Q. Okay. So you would never have seen this paper by Dean Baker where he goes through all the limitations on why fiber burden analysis isn't a good indicator of what someone was exposed to or whether the dose that matters is what they were exposed to. You can't comment on that.
A. Well, I -- as I said, I've not read it. But I would -- I think that a limitation and not identifying a fiber that we have now come to understand is not likely pathogenic, that's not particularly important.
MR. SCHACHTER: Your Honor, object to his displaying and cross examining on an article that he hasn't --
MR. FINCH: Okay. If the witness isn't familiar with it, I'll move on.

Q. You would agree with me that we've already said that the -- there are -- some of the limitations on fiber burden analysis, you're looking at the lung not the pleura, correct?
A. Right.
Q. You're looking at less than 1 percent of the lung tissue when you do a fiber burden analysis, correct?
A. Correct.
Q. You have to sample a portion of the lung so there are going to be different levels of asbestos fibers found in the lung depending on where the sample hits, correct?
A. Well, yeah, but for that reason we tend to use the peripheral lung where the asbestos fibers tend to be deposited.
Q. All right. We -- your 2002 article mentioned the microscope magnification/resolution issue, correct?
A. Yes.
Q. Meaning that your microscopes that you use at Duke don't see really thin fibers. By that I mean fibers less than 0.15 microns. You don't crank up the magnification high enough to see those thin fibers, right?
A. Yeah. Why would we? I mean, they're not pathogenic.
Q. There's also the issue of -- we talked about on direct the biopersistence of different fiber types, right?
A. Yes.
Q. That's a limitation on the use of what's in somebody's

lungs to make any kind of statement of what they were exposed to 30, 40, 50 years ago, correct?

A. Correct.

Q. You agree that chrysotile asbestos when breathed into the lungs can cause or contribute to a carcinogenic process prior to degrading.

A. No. And again, I agree that chrysotile mine dust with its natural contaminant can. This seems -- this particular slide that you're showing me seems to me to be a restatement of the -- what we call the hit and run process. The chrysotile is breathed into his lungs -- is breathed into an individual's lungs. Contributes to a carcinogenic process and it disappears. And for me I don't know of any scientific basis to support that.

MR. FINCH: Can you show the deposition of Dr. Sporn taken June 10, 2008, in a case called *Depiece versus Riley* (phonetic).

Q. Dr. Sporn, you were asked this question: "Do you have an opinion as to whether chrysotile asbestos when breathed into the lungs can cause or contribute to a carcinogenic process prior to degrading?

"Yes.

"And what is your opinion?

"It can.

"It can, is that what you said?

"Yes."

Is that your answer under oath in 2008?

A. That was.

Q. All right. You would agree that the world's scientific literature would treat this as a chrysotile cohort. Something that is -- predominant chrysotile exposure with potential or a little bit of amphibole exposure.

A. I'm sorry, you're going to have to --

Q. Sure. If there was a cohort of people that were exposed predominantly to chrysotile, 99 percent to chrysotile -- 95 percent to chrysotile, and there might have been some amphibole contamination, either through the tremolite in the ore or because there might have been some amphibole contamination in another part of the plant, the world's scientific literature in peer reviewed publications treats it as a chrysotile cohort.

A. I'm only aware of maybe two or three people that would -- cohorts that would fit into that definition.

Q. Okay. Are you familiar with this paper that was published in the British Journal of Cancer in 2012 called "Estimating the Asbestos-Related Lung Cancer Burden from Mesothelioma Mortality." The lead author is McCormack, and Julian Peto is one of the co-authors.

A. Yes.

Q. Okay. And they list in that paper chrysotile cohorts,

and they called them pure or predominant. And they go through 15 different studies and they treat those studies -- they call them chrysotile cohorts for the purposes of the analysis they are doing in this peer reviewed paper in the British Journal of Cancer, correct?

A. Yes.

Q. Okay.

A. But if you could go back to that slide, I would point out that I would disagree with number 20, the Balangero Mine. And I would disagree with the North Carolina textile plants which we have already been through in our discussion today.

Q. Okay. You would disagree with Julian Peto, and the other people, who is a world-renowned epidemiologist in calling this a -- these are chrysotile pure or predominantly chrysotile cohorts.

A. I would disagree with the -- actually, I would also disagree with him on number 21, the Quebec asbestos factory. That was not -- that was not a pure chrysotile plant. There was amphibole asbestos that was demonstrated in the lungs of patients with mesothelioma.

I would also disagree with 20. I would also -- the Balangaro Mine.

And I would also disagree with 27, the North Carolina textile plants in the USA.

Again, they're saying predominantly chrysotile. When

they say predominantly chrysotile, it sounds to me like there's a difference between predominantly chrysotile and pure chrysotile. I think in a predominantly chrysotile plant that there was an amphibole asbestos used in those facilities.

Q. And these are the cohort studies that IARC and the National Academy of Sciences and the United States NIOSH rely upon in doing risk assessments for the relative potency of the different fiber types. Those studies have been published in the peer reviewed literature.

A. I'm not sure they seek to establish fiber potency. But I think that they certainly do use these as a means for determining that chrysotile causes asbestos, which is why I think the IARC document is flawed.

Q. You just said chrysotile causes asbestos. I think you meant to say chrysotile causes mesothelioma.

A. I beg -- yeah, chrysotile mine dust causes mesothelioma.

Q. The world scientists outside of the courtroom debate, for whatever purposes they put it to, whether you think it's overly precautious or not, rely on these cohorts to make a conclusion that chrysotile causes mesothelioma.

A. Well, the world -- the world opinion is also -- IARC relies on the opinions of people like Infante and Phil Landrigan who are very much a part of the litigation.

Q. This was a letter to the editor published a couple of weeks ago by Dr. McCormack and Dr. Peto. And what they write

is "On the carcinogenicity of chrysotile, our article clearly shows that there are both excess of mesothelioma (four mesothelioma deaths per one thousand deaths) and lung cancer associated with chrysotile. This is entirely consistent with the IARC classification of chrysotile as a group 1 carcinogen to humans. At no point do we conclude that mesothelioma occurring in chrysotile exposed cohorts is due to other asbestos types; rather, we consider it valid to discuss that when multiple carcinogenic fibers are present. The relevant contribution of each is more difficult to disentangle. This is particularly the case for chrysotile in the presence of amphiboles because, as concluded by the most recent meeting of the IARC monographs, the latter appears to have a greater potency for the induction of mesothelioma than does chrysotile."

You're familiar with Dr. Peto's statement to that effect, correct?

A. Yes.

Q. Let's talk a little bit about fiber potency differences. You're aware that --

MR. SCHACHTER: Again, Your Honor, this goes beyond the direct examination. We didn't talk about potency differences at all.

THE COURT: All right. Go ahead.

MR. FINCH: He talks about it in his report.

THE COURT: Go ahead.
Q. You're aware that in 19 -- in 2008 the Environmental Protection Agency convened a science advisory board to look at a fiber potency model proposed by Berman and Crump?
A. Right.
Q. That estimated the relative potency of the various fiber types, crocidolite, amosite, chrysotile, correct? You're aware that happened.
A. Yes, I am.
Q. And you're aware that your colleague, Victor Roggli, wrote a letter on behalf of Caterpillar to the EPA science advisory board that says there is a big difference between the fiber types, correct?
A. Correct.
Q. And you're aware that the science advisory board included people like Julian Peto on it, correct?
A. I don't know who sat on the EPA on this regard.
Q. All right. You do know that the EPA convened a special panel of experts from around the world to analyze this question, right?
A. Yeah, and I know that they chose to ignore what was put forth, recommended to them by Berman and Crump.
Q. They chose to say -- okay, that's fine.
Ultimately, the EPA science advisory board concluded there wasn't enough historical exposure data to quantify the

differences between asbestos fiber varieties. Isn't that what they concluded?
A. That's sort of the summation of what they concluded.
Q. Okay. Now, the judge is going to hear testimony about projections of mesothelioma deaths done by doctors at Mt. Sinai in 1982 as a way to give him a basis to project the future number of asbestos claims in this case. You're familiar with Nicholson, Perkel, Selikoff, "Occupational "exposure to asbestos: projected mortality and population at risk," correct?
A. Yes.
Q. Okay. You're aware that Dr. Nicholson and Dr. Selikoff have basically gotten that projection right over the past 30 years, correct?
MR. SCHACHTER: Objection, beyond his area of expertise.
THE COURT: Well, he can answer it if he can.
A. I can't answer that.
Q. So you don't know whether -- you're certainly not going to dispute if other people come in here and testify that Nicholson got it right, you're not going to say he didn't get it right, correct?
A. No, I'm not -- I'm completely neutral in that regard.
Q. Okay. You recognize William Nicholson is an eminent scientist in the field of asbestos-related disease, correct?

A. I'm not familiar with much of his writings at all.
Q. You're not aware that Nicholson has published multiple papers on the epidemiology of asbestos-related diseases? You're not familiar with that?
A. I'm sure I've seen them. I can't -- I can't cite one offhand.
Q. Okay. So I guess -- are you familiar at all with Dr. Nicholson's paper, "The Carcinogenicity of Chrysotile Asbestos," a review that he published in 2001, one of the last things he published in the peer reviewed literature before he died?
A. I'm not aware of that.
Q. Don't you cite that paper in your rebuttal report in this case? Or at least you comment on Dr. Welch's citation to it.
A. I may have, yes.
Q. Okay. And what Dr. Nicholson, who 30 years ago projected mesothelioma deaths in the United States and got it right. What he wrote in 2001, "The case that chrysotile is a potent causative factor in producing mesothelioma is a strong one. It is shown to be so in a comparison of more than 40 studies of different fiber exposure circumstances.
"All available data suggests that it dominates the risk in those circumstances where it is the principal fiber used. The risk of chrysotile in producing mesothelioma is similar to that of amosite on a per fiber exposure basis."

He published this in the peer reviewed literature in 2001, right?
A. He may have, but he didn't get that right.
Q. Okay. You're saying he got that wrong.
Well, you're familiar -- we just talked about this paper from the British Journal of Cancer six months ago, right?
A. Right.
Q. Okay. There they are estimating asbestos-related lung cancer burden from mesothelioma mortality. And they've got chrysotile cohorts. They've got crocidolite cohorts. They've got three or four amosite cohorts. And they've got a lot of mixed cohorts. You're familiar with that paper, correct?
A. Yes.
Q. All right. And they have a table that compares mesothelioma deaths per 1,000 non-asbestos related deaths, right, Dr. Sporn?
A. That's what that table is.
Q. All right. And so the biggest rate of mesothelioma deaths per 1,000 non-asbestos related deaths is for crocidolite at 93.2, right? That's what you would expect.
A. Sure.
Q. Second biggest is mixed, right? Mixed fibers exposures is at 42 per 1,000, correct?
A. Yes.
Q. Then the next one down is amosite. That's at 18 per

1,000 non-asbestos related deaths, right?
A. Right.
Q. And then when you get to chrysotile, it's down to 4.1, right?
A. Right.
Q. Okay. So if you do the ratios, 93 to 18 to 4 is about like 100 to 20 to 4, right?
A. Close enough.
Q. Which is more like 25 to 5 to 1, right?
A. More or less, yes.
Q. All right. Latency period. You would agree with me that the median latency period for mesothelioma in your paper, the 1,445 paper, was on the order of 30, 35 years, right?
A. It's measured in decades, yes.
Q. Okay. But if I were to say 35 years is the median latency period for mesothelioma in the United States, you wouldn't quarrel with that.
A. No.
Q. Okay. And what that means, that means that half of the cases would have -- by latency, just so we're real clear. Latency is the time from the first exposure to asbestos until someone develops mesothelioma however many years later, correct?
A. Latency periods are the time from the first exposure to anything to the development of clinical disease, yes.

Q. So if we're talking about cigarette smoking, if you first started picking up cigarettes when you were 15 and you developed lung cancer at 65, that's a latency period of 50 years. That's what you mean by latency in the medical epidemiological context, right?
A. Right.
Q. Okay. So when you're talking about median latency, that means that half the people would have a latency -- in your group would have a latency shorter than 35 years and half of them would have it more than 35 years, correct?
A. Right.
Q. Okay. And although the median is 35 years, it can be -- there are cases in your case series where the first exposure was 50, 60 years before, correct?
A. Absolutely.
Q. Okay. And you would agree with me that when you're -- the people in your 1,445 case series, a lot of times they often don't know all the asbestos they may have been exposed to in their career or their life, correct?
A. Yes.
Q. And a lot of times the patient has got mesothelioma, patients you have treated with mesothelioma will say, yes, I worked around this kind of asbestos product, but they won't -- and they will have forgotten about or they won't have remembered that they were exposed to asbestos in the navy ten

years before, right?
A. They might not have even known.
Q. They might not even know. And it's only when you look in some of their lungs you say, hey, jeez, this guy was exposed to asbestos from pipe insulation in the navy; therefore, there was some exposure that he totally honestly with you didn't know anything about, right?
A. Sure.
Q. So if somebody says, you know, I don't know if I was exposed to asbestos from insulation in the navy, they're not lying. That's perfectly plausible, correct?
A. Sure.
Q. And you would agree with me on this concept of latency, if you were exposed to asbestos five years ago and you got mesothelioma, that's not within the latency period that you would expect to be an asbestos-associated mesothelioma, correct?
A. Latency periods of less than a decade have been described usually only in cases of massive exposure.
Q. And what I'm getting at is if the median latency period for mesothelioma is 35 years, which means half the cases would have latency periods shorter than that and half longer than that, if you only waited 30 years after a cohort of people was exposed, you wouldn't necessarily know if mesothelioma was going to develop in that cohort, correct?

A. No. There's a lot of things you don't know. I mean, you don't know who -- I mean, that's the big mystery with mesothelioma. We have thousands and thousands and thousands of people who have been exposed to asbestos and it's a rare disease. There are 3,000, 4,000 cases in our country a year and hundreds of thousands of people have been exposed. So I'm not sure what that means.

Q. What I'm getting at is, let's say you design a cohort study where you're looking to see whether exposure to a carcinogen causes a disease and you know the latency period, the average latency period for the disease causation is 30 years and you have a cohort of a thousand people and you expose them all to the same amount of stuff. If you only wait 20 years, you're not going -- it's not going to tell you anything really about whether or not that carcinogen can or cannot cause a disease, correct?

A. You don't know what --

Q. Okay.

A. You don't know what -- you don't know anything one way or another. You don't know that these folks are definitely going to come down with mesothelioma. You only can deal with the data that you have at hand.

Q. Okay.

A. And I would submit to you that, you know, to speculate beyond who might have got mesothelioma, that lacks scientific

foundation.
Q. I'm not trying to get you to speculate, Dr. Sporn.
Let me ask you this. Are you familiar with the preamble to the IARC 2012 monograph where they say that "it is important to note that evidence of lack of carcinogenicity obtained in this way from several epidemiological studies can apply only to the types of cancer studied, the dose levels reported and to the intervals between first exposure and the disease onset observed in these studies. Experience with human cancer indicates that the period from first exposure to the development of clinical cancer is sometimes longer than 20 years. Latent periods substantially shorter than 30 years cannot provide evidence for lack of carcinogenicity."
That's what IARC said in the preamble in 2012, correct?
A. That's what they've stated there.
Q. Okay. You don't disagree with that, correct?
A. Well, it's a very bland statement. It also -- I would also add if I was penning that, I would say latent periods substantially shorter than 30 years cannot provide evidence for lack of carcinogenicity nor can they provide meaningful information regarding the probability of the development of carcinogenicity 10, 20 years down the road.
Q. Isn't it true that none of the brake worker studies that Dr. Garabrant talked about had a latency period for all the people in the cohort longer -- all of them had longer than 30

years?
A. I don't remember what the latency periods were.
Q. All these scientific agencies have said --
A. But if you'll excuse me. The latency period also is inversely proportional to the degree of exposure.
Q. I'll get to that in a minute. What that means is the more asbestos you're exposed to, the shorter the duration for the latency period, correct?
A. Correct.
Q. So if you're exposed to a ton of asbestos, you might have a 20 year latency period; less asbestos exposure you might have 50 years?
A. If you get mesothelioma at all.
Q. If you get it at all, right.
A. But you may not. And the minority of people do get mesothelioma.

THE COURT: Let's take a break.
MR. FINCH: Okay.
THE COURT: Come back, I guess, at 10 after.
(Brief recess at 3:57 p.m.)

THOMAS SPORN

CROSS EXAMINATION (Cont'd.)

BY MR. FINCH:
Q. Good afternoon, again, Dr. Sporn.
A. Mr. Finch.

Q. I think I'm in about the 7th or 8th inning here, so we're going to get you out of here.

You would agree with me that all of these organizations have stated that there is no safe level of exposure to asbestos.

A. Yes.

Q. And they haven't made any qualification or exception for chrysotile asbestos in their statements, correct?

A. No, sir, they haven't.

Q. Are you familiar with the National Cancer Institute, if you go on the internet today they have something called a fact sheet relating to asbestos exposure and cancer risk. You Google that, National Cancer Institute fact sheet, you'll find that on the internet, right?

A. No doubt.

Q. And you're aware that the car companies wrote to the National Cancer Institute and sent them a bunch of epidemiological studies sometime in 2005 or 2006 and asked them to change the National Cancer Institute fact sheet, correct?

A. I'm not aware of that.

Q. Well, today the National Cancer Institute fact sheet states, "Studies evaluating the cancer risk experienced by automobile mechanics exposed to asbestos through brake repair are limited, but the overall evidence suggests there is no

safe level of asbestos exposure." And they cite to a couple of documents, correct?
A. That's what they say there.
Q. And then one of the things they cite to is the World Health Organization 1998 publication on chrysotile asbestos which is about 300 pages thick, correct?
A. I'm not sure about that. But as far as the statement you're displaying in front of me, I would submit to you that there are studies evaluating the cancer risk experienced by automobile mechanics exposed to asbestos in brake repair are not limited, but there are a number of them. And that the overall evidence suggests there is no safe level of asbestos exposure. Now are they talking about through individuals engaged in brake repair, in which case I would disagree. If they were talking about safe levels of asbestos exposure in general, that's a reasonable point.
Q. Let's talk about black spots. You're familiar with the Journal of Respiration?
A. Yes.
Q. Okay. This is an article published in the Journal of Respiration in the year 2002 that discusses the "Black Spots of the Parietal Pleura: Morphology and Formal Pathogenesis." Do you see that article?
A. Yes, I do.
Q. What they said is that black spots seen as

flat-to-nodular lesions of the parietal pleura are common findings in former miners. They represent areas of coal dust accumulation."

The conclusion -- and this is in the abstract of the paper. "Although there are hints for an increased proliferation of mesothelial cells in some areas with black spots, our findings do not support the classification of black spots as an obligate early lesion in the development of malignant mesotheliomas."

Do you see that?

A. I do. And I don't know if you care to ask me what I think of this, but I think this is -- it is an article that flies in the face of something that I referred to in my report by Dr. Boutin and colleagues where black spots seen as flat-to-nodular lesions of the parietal plural are common findings not just in former miners, but they are common findings in the population at large and people who live in areas where we breathe a lot of soot and automobile exhaust, they correspond to stomas. And that's where amphiboles congregate.

And I would disagree, although I have not read this paper, I would disagree with the -- "although there are hints for an increased proliferation of mesothelial cells in some areas with black spots, our findings do not support the classification of black spots as an early lesion in the

development of mesotheliomas."

I don't think that's what Boutin and colleagues were saying. I think they were saying -- and their bottom line in their paper is that's why mesotheliomas begin in the parietal pleura and not in the visceral pleura of the lung.

So I think this is all well and good, but I don't think that this changes my opinions regarding the translocation of amphibole fibers to the pleura nor the cite of development of malignant mesothelioma in the parietal pleura of the chest wall.

Q. Let me see if I can boil all that down. You would agree that this article is inconsistent with Boutin's conclusion about the significance of black spots.

A. Well, I don't think Boutin was trying to say that the black spots were precursor lesions, which they refer to as obligate early lesions in the development of mesotheliomas. All that Boutin was saying is the commercial amphibole fibers are concentrated in the black spots in the parietal pleura. Parietal pleura is where the vast majority of mesotheliomas develop and that's a potential explanation for why.

Q. A potential explanation.

A. Well, a very good explanation why.

Q. But you would agree with me there are scientists who differ with you about the significance of black spots in the parietal pleura.

A. Well, this is the first one I've seen and -- I know a great many pathologists from Germany and I don't know any of these individuals and so I'm not sure what their experience with mesothelioma is so -- I mean, the paper is what it is, but this doesn't, I don't think, change the contravening wisdom regarding development of --

Q. It was -- it was published in a peer reviewed journal called Respiration, correct?

A. Yes.

Q. All right. You talked a little bit about exposure to asbestos from ambient air. I just want to see if you'll agree with some basic third grade math.

You would agree a reasonable approximation, if somebody is working, how many breaths of air they take in a minute is anywhere between 12 and 20 breaths. Probably around 16 if you're an adult man and you're not some four minute miler or something, right?

A. Yeah, I mean, tidal respiration is 12 to 16 breaths per minute.

Q. And one breath of air is approximately 500 cubic centimeters that go into the lung that is not recycled air but new air into your lung.

A. Tidal -- your tidal volume, your tidal breathing is based probably about 10 CCs per kilo body weight. So 500 to 700 CCs.

Q. 500 CCs for a man, a grown man that's my size. 500 CCs. This is a water bottle, that's 500 CCs of water. 500 milliliters of water is the same volume of water as CCs of air.
A. That's more like 250 to me. Is that what it says? Does it say 500?
Q. It says 500. So you would agree with me that one breath is 500 cubic centimeters of air, right?
A. Yes.
Q. Okay. And that if you breathe 16 breaths a minute and there's 500 cubic centimeters of air for each breath and you're in an environment where there is one asbestos fiber per cubic centimeter, that means you're breathing 8,000 fibers in a minute, right?
A. Seems right.
Q. Okay. And just so we get on the record what a cubic centimeter is, a cubic centimeter is about the size of a sugar cube. It's a centimeter by a centimeter by a centimeter, right?
A. That's the example I was going to give.
Q. Okay. And so if it's 8,000 fibers in a minute, it's 120,000 fibers in 15 minutes at one fiber CC, right? Third grade math. Agree with that?
A. Yes.
Q. And it's 480,000 fibers in an hour, right?

A. Correct.
Q. And it's a little less than 4 million fibers of asbestos breathed in one working day if you're in an environment with one fiber per cubic centimeter, correct?
A. Right.
Q. Now, are you familiar that there are various estimates of the exposure -- of the amount of asbestos found in the background ambient air, correct?
A. Correct.
Q. I'm going to put up one from the ATSDR in 2001 in just a second which is a little bit lower than what you have in your textbook, but you've got a page in your asbestos-associated diseases book that has a table that summarizes asbestos exposure samples in different environments, right?
A. Yes.
Q. And you know, I'll show it to you in a minute, but I'm going to do this with the ATSDR because it makes the math easier, frankly. While I can do math quick in my head, I sometimes -- anybody can make a mistake without a calculator, right? You would agree that anybody can make a mistake with math sometimes.
A. Well, I sure have.
Q. Huh?
A. I sure have.
Q. So just because you're not perfect with math doesn't mean

that your conclusions aren't valid, correct?
A. Right.
Q. So in your book, the median fiber per CC concentration for the air of 48 United States cities is .00005. Four zeros and a five, is what it says in your book at page 26. Right?
A. I'd have to see it.
MR. FINCH: Can I approach the witness, Your Honor?
A. Yes, that's the concentration of fibers per CC, yes.
Q. And it's -- and it's what I just said it was which is zero point four zeros and then a five, right?
A. Right.
Q. Now, you're aware that the ATSDR, which is the Agency for Toxic Substances and Disease Registry, they published a big, fat book on asbestos and disease in about 2001, right?
A. Yeah.
Q. And they stated that since there are 1 million cubic centimeters in a cubic meter, there typically would be 0.00001 fibers per milliliter of asbestos in air in rural areas. Typical levels found in cities are about 10-fold higher.
And I'm going to start with their number for rural areas and then I'm going to use your number out of your book for the cities just so the math is easy.
If somebody is resting, they're going to be breathing a little bit less maybe than if they are working, right? Little bit less frequently. Would you agree with that?

A. Yeah, the minute ventilation if they are resting would be less than if they weren't.
Q. Okay. So 12 breaths a minute if you are just doing nothing is a reasonable estimate of how many times you breathe a minute, right?
A. Yes.
Q. And as we established, one breath is 500 CCs of air. That may be a little bit on the low side for a grown man, but that's a reasonable estimate of air per breath, right?
A. Yes.
Q. And again, 12 breaths at 500 CC of air times 6,000 CCs in a minute, that's 6,000 cubic centimeters of air in a minute. You would agree with that, right?
There's 360,000 cubic centimeters of air in an hour. That's just multiplying 6,000 by 60. That gets you to 360,000, right?
A. Yes.
Q. And if you do that, since you've got to breathe 24 hours a day or you're going to die, that gets you to 8,640,000 cubic centimeters of air breathed in a day. That's a reasonable estimate of how much cubic centimeters of air someone breathes in a day, right?
A. Seems reasonable.
Q. Okay. And if the fiber concentration in the air, the asbestos fiber concentration is .00001. If you do that math,

you get 86 fibers in a day at rural ambient air concentration, correct?
A. Yes.
Q. All right. So -- and this is the page out of your book I just showed you, right, with the air of 48 U.S. cities, median concentration is .00005. See that, Dr. Sporn?
A. Yes.
Q. Okay. So if in the rural air, if you breathe in 86 fibers a day, in the cities' ambient asbestos exposure, all you have to do to figure out what the difference is between the rural and cities is multiply by 5, right?
A. Owing to the higher concentration.
Q. In the cities.
A. Right.
Q. So ambient air exposure, whatever people are breathing -- and these are studies based on what the ambient air exposure was 10, 20 years ago, correct?
A. Yes.
Q. And you would agree with me that as we have used less asbestos in the world or in the United States, then you would expect the ambient air concentrations to go down over time?
A. I don't know if that's necessarily true because most -- because we're still finding commercial amphiboles in people's lungs and those have been out of the marketplace for a number of years. So I don't know.

Q. Okay. All right. So let's just say that it's the same now as it was ten years ago. The ambient air concentration is 432 asbestos fibers in a day if you are in a city, right? That's a reasonable estimate.
A. Seems to be based on your math.
Q. As compared to 3.8 million fibers in one working day at one fiber per CC, correct?
A. At one fiber per CC -- yeah.
Q. Okay. And at 0.1 fibers per CC, and I actually didn't do the math right here because if you divide -- all you have to do to figure out how many fibers you breathe at the current OSHA permissible exposure limit of 0.1 fiber per CC is to divide that 3.8 million figure by 10, right?
A. Right.
Q. And so one day of working in an environment with 0.1 fibers per cubic centimeter of asbestos is 364,000 -- or 384,000 a day versus 432 fibers in ambient air, right?
A. Yes.
Q. All right. And so you could get to in a couple of weeks working at 0.1 fiber per CC asbestos, you could get to as many fibers as you breathe in ambient air in your whole life, right? If you did the basic math.
A. Again, that would depend on what size fibers they were and what length they were, where they were deposited. If they were -- if they were less than half -- less than 5 microns, if

they were long, curly fibers greater than 30 -- it's hard -- it's hard to know with those raw numbers how many of those asbestos fibers will be deposited in the parts of the lung that are involved in gas exchange.

Q. I'm just asking about what comes into your lung. And you --

A. No --

Q. And the way historically they measure asbestos fiber in the air, you would only measure fibers longer than 5 microns for fibers in the air, right? Do you agree with that? Historically they're only measuring fibers longer than 5 microns.

A. Okay.

Q. Now, you're familiar with that document, the Helsinki criteria?

A. Sure.

Q. Your colleague Victor Roggli was a contributor to it, correct?

A. Sure.

Q. That was a document, it was a group of -- multidisciplinary gathering of pathologists, radiologists, occupational and pulmonary physicians, epidemiologists, toxicologists, industrial hygienists, and clinical and laboratory scientists specializing in tissue fiber analysis. Collectively, the group has published over a thousand articles

on asbestos and asbestos-associated diseases. That article was published in 1997, correct?

A. Correct.

Q. And what they state in the first page is, "In general, reliable work histories provide the most practical and useful measure of occupational asbestos exposure."

Dr. Roggli agreed with that in 1997, correct?

A. That's what it says in Helsinki. But again, it's a big document and it is -- and it is not one that I don't believe from my perusal of it to speak to causation of fiber type.

Q. Well, doesn't the document actually say all types of malignant mesothelioma can be induced by asbestos, with the amphiboles showing greater carcinogenic potency than chrysotile?

A. Well, again, I would agree with that statement with the caveat that, again, you have to -- when you're talking about chrysotile, you cannot frame any type of discussion about chrysotile without breaking it down. Is it chrysotile fibers you're dealing with? Is it chrysotile dust you're dealing with? Is it chrysotile ore? Is it chrysotile dust from mines?

Q. This document was published in the medical literature and it states that "an occupational history of brief or low level exposure should be considered sufficient for mesothelioma to be designated occupationally related to asbestos exposure.

"A history of significant occupational, domestic or environmental exposure to asbestos will suffice for attribution."

That's what the Helsinkey criteria says, correct?

A. It does. But I would submit to you that a -- someone who is, let's say for argument purposes, a shade-tree mechanic, that those -- the people who penned the Helsinki criteria would not look upon that as significant occupational exposure.

Q. And there is no qualification in those statements about, oh, this doesn't count for chrysotile, this doesn't count for brakes, correct?

A. No.

Q. All right. Now, you talked a lot about your work with Dr. Roggli, and you and Dr. Roggli have worked together for what, almost 20 years?

A. Yes.

Q. You know that Dr. Roggli has testified on multiple occasions in asbestos litigation, correct?

A. Yes.

Q. Let me show you a sworn statement that Dr. Roggli testified to.

Dr. Roggli lives in the county of Durham, right?

A. He does. Been to his house.

Q. "My name is Victor Roggli. I am over the age of 18 years and fully competent to make this affidavit. The facts stated

in this affidavit are within my personal knowledge and are true and correct."

You wouldn't expect Dr. Roggli to lie about something, would you?

A. No, I wouldn't expect him to lie about anything.

Q. "While there is no question among experts in the field that chrysotile dust causes the disease mesothelioma, there is some dispute regarding whether it is the chrysotile itself or the tremolite contaminant of the chrysotile dust or both that causes the mesothelioma. The resolution of that dispute is, however, purely an academic exercise. Over 99 percent of the chrysotile used in the United States was mined in Canada. Every published study addressing the issue indicates that there is some degree of tremolite contamination in all chrysotile ore mined in Canada. Further, there is no evidence that the tremolite contamination is removed during the processing of the chrysotile ore."

That's what Dr. Roggli swore to in this affidavit, correct?

A. Can you tell me when the affidavit was.

Q. The affidavit was in a case called *Innerarity* and it was in 2001. Let me just see if you agree that Dr. Roggli's opinions in 2001 are consistent with the affidavit.

"The scientific and medical community has yet to determine the level of exposure to asbestos below which

mesothelioma will not occur. While there is no threshold, there is insufficient evidence to implicate levels of asbestos exposure, exposure to asbestos that occur as a result of background or ambient air exposure. Very low levels of exposure above background, however, have been demonstrated to cause mesothelioma.

"It is also my opinion that it is the total dose of asbestos, regardless of fiber type, that the patient experiences that causes the disease.

It is further my opinion that each and every exposure to asbestos that an individual with mesothelioma experienced in excess of a background level is a substantial contributing factor in the development of the disease."

I read that correctly, correct?

A. Yes.

Q. And substantial contributing factor, that's not a concept that you and Dr. Roggli or any doctor talks about in the real world of seeing patients. That's a question you get asked in front of a jury in the context of a legal case, correct? It's a mixed question of medical opinion, but doctors don't go around saying in my opinion smoking was a substantial contributing factor in causing this man's lung cancer. They say his lung cancer was caused by smoking, right?

A. Ordinarily when I issue a diagnosis of mesothelioma, I don't opine as to the cause one way or another unless I'm

specifically asked to.
Q. And here Dr. Roggli was specifically asked and he was specifically asked in the context of a case involving Boyce Innerarity, "I have reviewed Boyce" -- I can't pronounce the gentleman's name -- "Innerarity's deposition testimony regarding his work history. Based on his testimony, it is my opinion that his exposure to insulation materials containing asbestos was not the sole cause of his disease. He was exposed to many different products that contained asbestos. Specifically, I reviewed Mr. Innerarity's testimony regarding the manner in which he removed Garlock asbestos-containing gaskets, and to the extent that on repeated occasions over a career spanning three decades Mr. Innerarity as a regular part of his job removed dry, adhered Garlock gaskets containing up to 85 percent chrysotile asbestos from pipe flanges using electric wire brushes and wore no respiratory protection, and to the extent that such activity created dust levels substantially above background, it is my opinion that exposure to dust from the Garlock gaskets was a substantial contributing cause in the development of his mesothelioma."
That's what Dr. Roggli wrote in an affidavit he signed in 2001, correct?
A. Yes. And was there a question to me there or just that Dr. Roggli wrote that affidavit?
Q. Yeah, Dr. Roggli wrote that affidavit in 2001.

A. He wrote that affidavit, yeah.
Q. Okay. Now, you talked about Dr. Hammar on direct, correct?
A. Yes.
Q. He was a mentor of yours when you were out in Washington state, correct?
A. Yes.
Q. And you know that Dr. Hammar has published numerous articles in the peer reviewed literature on the causation and diagnosis of asbestos-related diseases, correct?
A. Yes.
Q. This textbook, "Dail and Hammar's Pulmonary Pathology" is a well-regarded pathology textbook, correct?
A. Yes. I wrote the chapter on occupational lung disease in it.
Q. Okay. There's also a chapter that Dr. Hammar wrote entitled "Neoplasms of the Pleura." You know that that chapter is in the book, correct?
A. Yes.
Q. Okay. And what Dr. Hammar writes is "No lower (minimum) threshold level of exposure to asbestos has been delineated below which there is no increase in the risk of malignant mesothelioma. And most authorities approach causation of mesothelioma by asbestos from the perspective of a no threshold model. One factor that emerges from the Peto model

and its modifications is that where there are multiple asbestos exposures, each contributes to cumulative exposure and hence to the risk and causation of malignant mesothelioma within an appropriate latency interval."

That's what Dr. Hammar wrote in his pathology textbook that was published in 2008 and is on the library shelves of many practicing pathologists around the country, correct?

A. Yes.

Q. Finally --

A. And am I just supposed to agree that that's out there or was there a question?

Q. You agree that that's out there, correct?

A. I agree that that's out there.

Q. Okay. And you agree that Dr. Hammar is a well-respected, well-regarded scientist in the field of asbestos and disease causation.

A. I would agree with that. He's also one that both Dr. Roggli and I find myself in significant and substantial disagreement on any number of cases, especially in the area of litigation.

Q. Okay. Let's talk about something I think -- this is my last topic and I don't think you're going to disagree with anything I say or anything any other doctors say.

Mesothelioma is almost invariably a fatal disease, correct?

A. Unfortunately, yes.
Q. And what often happens is -- one of the first things that happens is people get diagnosed with mesothelioma or they think they might have mesothelioma and there wil be a pleural effusion or some other kind of change that's causing them pain, right?
A. The pleural effusion might cause shortness of breath. It rarely causes pain. But they usually present with pains referable to the mesothelioma in their chest.
Q. And oftentimes they have fluid drained from their body. I've seen -- I've had client after client in the medical records, two liters of fluid taken out of the body. That's one of the -- that's oftentimes a consequence of having mesothelioma, correct?
A. Yes. And I personally have drained over liters and liters and liters and liters of fluid off the chest of people with mesothelioma.
Q. And then as mesothelioma progresses, it gets -- it causes a constricting of people's ability to breathe in many cases, correct?
A. Yeah. It causes breathing impairments due to reduction of lung volume, yes.
Q. And it causes people to undergo severe debilitating pain in many cases, right?
A. It's a terrible disease.

Q. It's a terrible disease. And the survival rate for people with pleural mesothelioma, most of them are dead within a year or two, right?
A. Fortunately that's changing, but overall historically the prognosis is measured in months to a few years.
Q. And in some people, if they're really young and in good shape, they'll try desperately to stay alive by having one of their lungs removed in a procedure called an extrapleural pneumonectomy where they take out the lung where the pleura is that has the mesothelioma in an attempt to extend their life, correct?
A. Yes. And to reduce pain, yes.
Q. And that's a procedure that you know that's done in New York, it's done in Philadelphia, it's done by Dr. Sugarbaker in Boston, correct?
A. And probably most of all at my hospital.
Q. And at Duke. I wasn't meaning to slight Duke.
And most of the time people with mesothelioma, even if they have that surgery, the tumor comes back and they die from it, correct?
A. Unfortunately.
Q. And they die in tremendous pain, correct? Many, many people with mesothelioma.
A. It's a terrible disease.
MR. FINCH: That's all I have, Your Honor.

THE COURT: Anything else, Mr. Schachter?
MR. GUY: Your Honor...
THE COURT: I'm sorry. Mr. Guy. I forgot about you.
MR. GUY: That's okay. As long as you don't forget about me completely.

CROSS EXAMINATION

BY MR. GUY:

Q. Dr. Sporn, my name is Jonathan Guy. I represent the FCR in this case.
A. Good morning -- or afternoon.
Q. You wrote your article about malignant mesothelioma and occupational exposure to asbestos in 2002, correct?
A. Yes.
Q. And was that published so it was available to the public?
A. Yes.
Q. Would it have been available to asbestos defendants like Garlock?
A. Sure.
Q. And your book, when did you first write your book?
A. That was published in 2004.
Q. And that's also obviously available to the public?
A. You can get it on Amazon.
Q. Is there any reason why Garlock wouldn't be able to consider the position that you're taking and have taken

concerning chrysotile or mesothelioma in evaluating whether to litigate and settle cases?
A. That is a decision that law firms and/or commercial firms make and I have no opinion in that regard.
Q. When you first wrote your article, you were at Duke, correct?
A. Yes.
Q. And you could have been reached by Garlock over the telephone quite easily, correct?
A. By telephone I'm hard to reach. Maybe by email.
MR. GUY: No further questions, Your Honor.
THE COURT: Mr. Schachter.

REDIRECT EXAMINATION

BY MR. SCHACHTER:
Q. Sir, you were asked some questions about various agencies that have looked at the mining studies that you told us about on direct, and looked at other studies and opined that chrysotile can cause mesothelioma based on those situations. Is that inconsistent with your opinion as long as we understand what chrysotile --
A. Yes. I think that -- whenever I enter into a discussion regarding the potential for chrysotile to cause mesothelioma, I want to make sure that the parties to the discussion, that we're all on the same footing on what we're talking about. Are we talking about chrysotile that's in the dust of mines

and chrysotile producing areas in Quebec? Are we talking about the chrysotile that was used historically in friction products? What are we talking about? And we need to be very, very specific about it in our discussion in that regard.

Q. Now, on your opinion on -- you were asked some questions about no safe level and I don't think you were able to fully explain. Has science yet established at what level the danger of asbestos related mesothelioma aises, what level of exposure creates the danger?

A. Not for the amphibole forms of asbestos. But if you -- if you look to what has been published in the literature regarding -- regarding chrysotile -- and this is chrysotile dust, dust from mines, or dust that has been liberated in areas where there are a lot of mines such as in Thetford, Quebec, those -- again, there is no, quote, safe level, but there's been a range of levels that have been observed and those are in the hundreds of fibers -- of fiber years.

Q. And if for regulatory purposes an entity wants to assume equal potency, does that mean that for a scientific fact the fibers on a fiber per fiber basis are equally potent?

A. No. For a public health model, they would take things -- I would assume like they do in other models, take things back to zero. The risk begins with that very first fiber. That's a separate argument from a scientific concern.

Q. Now, you're acquainted with Dr. Roggli, of course, right?

A. Yes.
Q. And you were shown an affidavit from 12 years ago or 13 years ago purportedly in a case.
A. Yes.
Q. Has he been testifying to the fact that low dose products like chrysotile based on the current science are not a cause of mesothelioma?
A. Yes, he has.
Q. All right. And when it comes to gaskets, you were also shown -- you were also -- they talked to you a lot about Mr. Nicholson or Dr. Nicholson.

Can you put up the exhibit.

You're familiar with William Nicholson. And he was discussed in -- he was about -- he was asked about whether gasketting work created a risk of mesothelioma, and prepared an affidavit opining that he "would conclude that the relative contribution of gasketting work to cancer, including mesothelioma, originating from workplace exposures is minuscule. Furthermore, the contribution to any non-malignant asbestotic disablement is literally nonexistent. From the data that exists from clinical findings of asbestosis exposed workers, it is clear that the exposure from installation or removal of gasket materials would not lead to any discernable disablement among those undertaking such work or among those exposed indirectly."

That was Dr. Nicholson in 1993 -- 83.

Is there anything inconsistent in what Dr. Nicholson was saying with the opinions that you've expressed here today?

A. No, sir.

Q. And based upon the current science, is our gaskets a cause of mesothelioma in human beings?

A. No, sir.

MR. SCHACHTER: Thank you.

THE COURT: You can step down. Thank you, Dr. Sporn.

THE WITNESS: Thank you, Your Honor.

(Witness stepped down.)

THE COURT: Do you have another witness you want to start today?

MR. SCHACHTER: Mr. Harris may.

MR. HARRIS: Yes, Your Honor.

THE COURT: Okay.

MR. HARRIS: At this time we call -- Garlock calls Larry Liukonen.

LARRY RAY LIUKONEN,

being first duly sworn, was examined and testified as follows:

DIRECT EXAMINATION

BY MR. HARRIS:

Q. Please tell us your name.

A. My name is Larry Ray Liukonen.

Q. Where are you from?
A. I'm from Burleson, Texas.
Q. What do you do for work?
A. I'm an industrial hygiene consultant.
Q. Are you a certified industrial hygienist?
A. Yes, I am. I have been since 1976.
Q. This case involves individuals who allege worked with asbestos gaskets and packings. Have you studied potential exposures associated with that work?
A. Particularly I've studied exposure with work with gaskets.
Q. We've heard about a study on gaskets by the United States Navy in 1978. Are you familiar with it?
A. Yes, I am.
Q. How so?
A. I was the lead author.
Q. What was the purpose of your study?
A. In 1978 the navy at least knew -- we didn't always do it, but at least we knew what we should do with friable thermal insulation products. So someone was wondering if they should look at other more traditionally non-dusty products and see if any modifications needed to be made to work procedures.
So they asked all of the industrial hygienists in the navy if anyone had looked at it and everyone replied, no, we really haven't taken a look at it. So they asked us in

Bremerton to design a study to evaluate all aspects of the life cycle of a gasket.

Q. When you were with the U.S. Navy, did you also research and assesss exposures to insulation used on ships?

A. Yes, I did.

Q. Have you brought with you documents from your work in the navy in the 1970s and the work of others that you have relied upon?

A. Yes.

Q. And have you prepared some slides to help us understand what the potential exposures were historically from working with asbestos gaskets and packing and insulation products?

A. Yes, I have.

Q. Before I ask you about that research, I would like to ask you a few questions about what qualifies you to discuss these matters. What was your educational background?

A. I have a bachelor of science in the biological sciences from the University of Minnesota. After that I went directly to graduate school and I received a master of science in environmental health where I studied air pollution and industrial hygiene.

Q. At this time it would probably be a good idea if you told us what industrial hygiene was.

A. Sure. Basically, what we do is try and prevent occupational diseases. We spend a lot of time in the

workplace looking at what people do, what chemicals or materials they work with, what sort of work practices they use. We -- if necessary we make measurements or we collect air samples or possibly noise measurements. We compare those results to recommended standards; and if necessary, we recommend corrective action.

Q. In your graduate studies, did you study asbestos or potential asbestos exposures in the workplace?

A. Yes.

Q. After you received your degree, where did you go to work?

A. After my -- I received my master's degree, I accepted a commission in the United States Navy and I did industrial hygiene for them on active duty for three years.

Q. All right. It indicates here from 1972 to 75 you were with the U.S., is it naval reserve?

A. I was in the naval reserve, active duty and naval reserve.

Q. Why did you join the navy?

A. When I went to -- when I was in my graduate program, several of my co-students were being sent there by the military. Military was sending people back to get their master's degree in industrial hygiene. Military was actively looking for industrial hygienists, so I thought that was a good opportunity.

Q. So you were actually in the navy. Did you have a rank in

the navy?
A. Yes, I did. I started out as lieutenant junior grade, lieutenant JG, and I finished as a lieutenant commander.
Q. It says here you were in Cincinnati; is that correct?
A. That's correct.
Q. What was the nature of that work?
A. We were basically a consulting group. We had occupational health physicians, we had occupational nurses and industrial hygienists. And we provided assistance to naval facilities primarily in the U.S., but some worldwide. When they needed special assistance for a project, either they didn't have the expertise themselves or maybe they didn't have enough staff, then we would go and assist with those projects.
Q. And you did that for three years; is that correct?
A. I did, yes.
Q. And then you left the navy; is that correct?
A. I left active duty.
Q. While you were there from '72 to '75, did you have any contact with asbestos products or asbestos that's used in the navy?
A. Not very much. The only one that I specifically recall is I was sent to a shipyard that was having a little problem with a ventilation system that they were using for asbestos fabrication. They needed somebody to come and take a look at it. And two of us went down there and helped with that

project.
Q. It looks like in 1975 you then left and went to Naval Regional Medical Center at Bremerton, Washington. Now, were you in the navy at this point?
A. No, then I was a civilian.
Q. So why did you go to Bremerton? You're out of the navy now, why did you decide to go back and work for the navy as a civilian?
A. The -- because the -- the really good industrial hygiene jobs in my opinion were at the shipyards and so I wanted to go -- number one, I wanted to go to one of the places that had a shipyard. And I picked Bremerton as my first choice because they had a reputation of having the best industrial hygiene program in the navy. So that's where I wanted to go.
Q. In 1976 we saw on the prior slide that you became a certified industrial hygienist.
A. That's correct.
Q. Why did it take you so long to become a certified industrial hygienist?
A. They require that you have five years of professional experience before you can take the examination. When you have an advanced degree, you can knock off one year, which is what I did.
Q. When you got to Bremerton, what were the industrial hygiene concerns there?

A. We had a lot of concerns because we had a ten thousand person shipyard. We covered the whole Pacific Northwest. We had air stations and ammunition facilities and lots of things. Our main concerns were asbestos from friable thermal insulation. We had concerns with heavy metals like cadmium and lead. We had concerns with noise. We had concerns with solvents.

Q. And what were the asbestos concerns specifically?

A. That would be rip out -- removal of thermal insulation aboard ship when ships came in for overhaul.

Q. You mentioned rip out. Tell us what rip out is.

A. Rip out is removal. It's the thermal insulation and -- when a ship comes in for overhaul, there's a lot of that that's removed and it can be very dusty. And it was something we were very concerned about.

Q. Had the asbestos controls been implemented at Puget Sound Naval Shipyard when you arrived or before you arrived in 1975?

A. There were some controls that were in place. Not very -- pretty primitive by today's standards. And we had a difficult time enforcing even the minimal controls that we did have. We did not get a lot of cooperation from some of the supervisors; therefore, not a lot of cooperation from some of the workers.

Q. When you say minimal controls, what type of controls are we talking about?

A. Well, theoretically they were to wet the material when

they removed it. Often didn't happen.

In terms of keeping people out of the area, it was simply a rope and a sign.

And you could work within 5 feet of someone removing thermal insulation without any protective equipment whatsoever.

Q. A rope. What about plastic sheeting and creating a tent or enclosure, was that going on in 1975 in Puget Sound?

A. It was not.

Q. Did you conduct any air monitoring during the thermal insulation removal while you were there?

A. Yes.

Q. What type of exposures did you find even during this period of minimal controls?

A. Our goal, if everybody did everything perfectly, our goal was to stable .02 fibers per CC. That usually didn't happen, and we measured 5, 10, 20 commonly and levels over a hundred and even over 200 fibers per CC.

Q. Okay. To put that in perspective, the OSHA permissible exposure limits in 1975 would have been the original one of five fibers per CC?

A. That's correct.

Q. Now, were there any controls in place when you arrived at Bremerton with respect to doing any work with gaskets and packing?

A. No, there were no controls whatsoever for gaskets or packing.
Q. And how long did you stay with the navy as a civilian industrial hygienist?
A. Four years.
Q. That takes us to 1979, I believe. And then from there you worked for the railroad for almost ten years or eight years.
A. Eight and a half years.
Q. What type of work did you do there?
A. I was the first industrial hygienist at that company and did a lot of noise monitoring, sampling for diesel exhaust, again, heavy metals, silica. Asbestos was a pretty minor concern there.
Q. And then in, I believe it was 1987, you formed a company with a partner, and then in '88 you formed your current company; is that correct?
A. That's correct.
Q. And that's Technical Safety and Health Consulting, Inc.
A. That's correct.
Q. And what type of work have you done since then?
A. We do comprehensive industrial hygiene. We've remained -- done a lot of it in the railroad industry. Do a lot of work for the railroad industry. A lot of routine air sampling and noise monitoring. I've been involved in

litigation-related activities the entire time, more so towards the end than I was at the beginning.

MR. HARRIS: At this time, Your Honor, we tender Mr. Liukonen as an expert in industrial hygiene.

MR. GEORGE: No objection, Your Honor.

THE COURT: All right. He'll be admitted as such.

Q. Mr. Liukonen, you've also published in the peer reviewed literature; is that correct?

A. That's correct.

Q. It looks like we've got two articles here. Can you tell us what those were.

A. Yes. The first one is an article -- a study that we did on exposure of railroad train crews to particulates from diesel engines, such as locomotives is what we were looking at.

The second one is a study I did with Dr. Weir which was regarding asbestos exposure from working with a diesel engine, removing the gasket material from a diesel engine.

Q. What type of gasket material? Were these special engine gaskets?

A. Well, they're basically the same type of gaskets you'd find everywhere else. There was varying percentages of asbestos. I think like some were as low as 5 percent. Others were probably as high as 75 or 80 percent asbestos.

Q. Were they compressed asbestos sheet gaskets?

A. Yes, they were.
Q. I would like to ask you now about the study that you did for the navy. This was in 1978; is that correct?
A. That's correct.
Q. There you are as L.R. Liukonen as the first author.
A. That's correct.
Q. The second author is Kenneth Still. And you understand that he's been designated as an expert witness in this case by Garlock; is that correct?
A. Yes.
Q. And then the third author is R.R. Beckett, and you understand that he has been designated as an expert witness by the committee; is that correct?
A. Yes, I do.
Q. You've read his report?
A. Yes, I have.
Q. And you've read his deposition; is that correct?
A. Yes.
Q. I believe you told us generally how this study came about. Could you tell -- remind me again exactly why you did this study.
A. The navy wanted to know at this point whether they needed to modify any procedures to keep exposures below the PEL at the time. And also there was -- the navy had a medical monitoring program that began at .1 fibers per CC. So another

goal of the study was to determine if people that worked with asbestos gaskets needed to be enrolled in this medical monitoring program.

Q. Was the .1 fibers per CC an 8-hour time-weighted average?

A. It really was, but as industrial hygienists generally, we tend to be very conservative and tend to think that -- we tend to -- we tend to think in terms of the exposures for short-term even though it really is a time-weighted average.

Q. And at this point in time, the 8-hour time-weighted average in 1978 had been lowered to two fibers per CC; is that right?

A. That's correct.

Q. Had you studied potential exposures to asbestos-containing gaskets or packing in the shipyard before this study?

A. We had two samples that we had collected before this time.

Q. And what were those samples in relation to?

A. They were in relation to an operation where -- gaskets are actually manufactured in the shipyard. There's a large number of gaskets that would be used in the shipyard, so instead of everyone making their own, we had a couple of employees that that was their job was to stamp out gaskets. One of them used an automatic punch press. The other used a mallet with a punch to punch the bolt holes in the gaskets.

And that's where those two samples had been collected.
Q. There were only two workers that were doing that type of work?
A. That's correct.
Q. And how many other people would have been working or coming in contact with asbestos gaskets during this time in the shipyard?
A. Oh, probably thousands of other people would use asbestos-containing gaskets.
Q. The -- what type of exposures did you find for this manufacturing type operation?
A. When we did those two samples in 1975, we included this -- those in this study as well. We reported three fibers per CC for one sample and five fibers per CC for the other one.
Q. When you received those samples, did you take any action?
A. We did. We recommended that they be included in the medical monitoring program and we recommended that that area be thoroughly cleaned.
Q. Why hadn't a report been done on gasket operations before your study?
A. Well, as an industrial hygienist I didn't really consider it as a real potential for exposure. Gaskets are encapsulated materials. They're not dusty materials. They're insignificant compared to the exposures that we would expect

from friable thermal insulation.
Q. Was sampling done under actual real world type conditions?
A. Absolutely. We collected them under normal work conditions in shops and aboard ship.
Q. Did the study have anything to do with litigation at the time?
A. No, none whatsoever.
Q. What operations did you study?
A. We studied all aspects. We tried to determine all aspects of the life cycle of a gasket.

Let me -- there we go.

We looked at storage, when the gasket materials are received and stored for use. We looked at the hand gasket punching; hand operated mechanical punching, which was basically pulling a lever down to punch a hole in the gasket; machine punching; hand shaping; machine sheering; machine nibbling; installation; removal with concurrent installation; removal and hand scraping; removal and wire brushing; and then clean up following removal. By clean up following removal, I don't mean cleaning the floor or something like that at the work area. I mean cleaning up the flange, the gasket remnants that are left on the flange.
Q. After the gasket is removed?
A. Yes.

Q. And that was a separate sampling that was done; is that right?
A. That's correct.
Q. And so these are -- are these all the operations involving gaskets in the shipyards that you could -- the shipyard that you could identify?
A. Yes.
Q. Which of these operations would be typical for someone that is working with gaskets, like a pipefitter or a machinist's mate -- a pipefitter in the shipyard or a machinist's mate in the navy?
A. They would typically be working with the -- here they're broken down. Would be the ones on the left side. This is the ones that most people would be working with. The ones that we consider as secondary manufacturer. Primary manufacturer to me is someone that actually makes the gasket sheet. Secondary manufacture is where they stamp the gasket out of that sheet.

Those are the processes on the right-hand side. There was a limited number of people in the shipyard that did that work, was probably two people full-time and another person part-time. Whereas the left side, that's the end user and that's -- we have large numbers of people to do those sorts of activities.
Q. So a company like Garlock would manufacture compressed asbestos sheet. It would get sold to or distributed to the

shipyard and then people -- at least two workers, I guess, in the shipyard would then cut from the sheets, cut out the gaskets that would then be used on the ships in the shipyard; is that correct?

A. That's correct.

Q. Okay. And you only had a couple of people there at the Bremerton shipyard doing that type of work; but on the left-hand side, the end user activities, how many workers were we talking about?

A. I would suspect thousands.

Q. Okay. So I want to focus mostly on those people for the purposes of this exam.

We've talked about fibers per CC in terms of exposure limits and so forth. Can you explain a little bit more about that process. How are the -- how are air samples collected?

A. I brought an air sampling pump here that we can demonstrate. We use -- it's a battery operated pump. We typically put it on the belt of the person. Then we use a flexible tube and then put a filter in what we call the breathing zone of the worker, as close as we can to where they breathe. Typically their lapel or collar.

Then we -- for asbestos, we take the cap off, which is unusual. We don't do that for very many materials. And then point it down so the dust doesn't fall on the filter; you're collecting samples that are in the air. And then you

actually -- you physically at some point when you're finished, you physically take the filter out of there and dissolve a portion of the filter and actually count the fibers under a microscope.

Back when we did the study in Bremerton, this is the current methodology. The only difference -- the main -- there are slight differences. The main difference in the method that we used back then, we used a larger filter. Our concentrations, our exposures were higher and a larger filter helped disperse the fibers better. When the concentrations started getting lower, we went to a smaller filter.

Q. Are there methods that tell you how to collect the sample and how to analyze the sample?

A. Yes, absolutely there are. It's a slightly different method now than it was then. We used 239. NIOSH is the one that establishes sampling in analytical methods like that. The current method is NIOSH 7400. Everyone must follow the same method, use the same techniques. Otherwise, you can't compare one sample -- a sample you collect and a sample someone else collected.

Q. And those methods, are they specified also in the regulations that actually set what the permissible exposure limits are?

A. Yes, OSHA also has a method essentially. It's the same as 7400 saying you must follow this method.

Q. Now, this was the methodology in 1975, 76, 77, 78, when you were with the navy and then at Bremerton; is that correct?
A. Yes.
Q. Was there an earlier methodology for collecting and analyzing air samples for asbestos purposes?
A. There was. Before this -- this light microscopy method we used -- where we actually count fibers, we used a method where you had a solution in a -- in what's called a midget impinger. You put 10 milliliters of solution, typically like distilled water, and then you would run air -- bubble air through that filter -- or excuse me, through that solution and you would analyze that solution. Actually count fibers and particles in that solution.
Q. And how were the results reported? Were they reported in fibers per CC, for example?
A. No, then they were reported as million particles per cubic foot.
Q. And that's abbreviated MPP --
A. CF.
Q. -- CF.
A. That's correct.
Q. When was that methodology used for air samples for asbestos operations versus the newer method that you described to what you used in your Bremerton study?
A. I think the conversion was really in the late -- late

'60s and early '70s. I took one of the first classes if not the first, I'm not sure, classes that NIOSH offered in sampling and analyzing asbestos dust and that was using the new method.

Q. When you did your -- back to the Bremerton study and the operations that you sampled. Were there control measures in place during the sampling for the different operations that you studied?

A. There were some control measures in place for some of the secondary manufacturing operations that we had put in place. There were no control measures in place for any of the end user activities.

Q. And so the control measures were on those operations on the right that were done by just the one or two workers in the shipyard.

A. That's correct, yeah. The only thing that could be remotely considered a control in the end user activity was for certain activities people, instead of throwing the debris on the floor, they would put it in a plastic bag for disposal.

Q. Okay. And what was that referred to in the report?

A. That was one of the housekeeping techniques we used.

Q. Any other controls for the end user activities?

A. No.

Q. Would the housekeeping controls, putting the scrap in a plastic bag, reduce the exposure a worker had for the

operation?
A. Putting it in a plastic bag wouldn't affect the exposure whatsoever.
Q. So what did you learn about the exposure from working with gaskets as an end user in the shipyard?
A. We learned that end users really didn't have exposure to asbestos from gaskets. That they were well below the OSHA standard. And for the most part, all of the samples were even below the level at which medical monitoring would begin.
Q. And so we've projected a slide here that identifies the different end user operations. I want to start here with the process.

So in storage, it looks like you evaluated a couple of different ways. And you collected 14 samples. Why would you collect so many samples on just storage of gaskets?
A. Well, we wanted to -- we wanted our study to be statistically significant. The navy asked that we conduct a statistically significant sample, so we tried to get as many as we could for different operations.

Storage, obviously, was something that was going on every day so it wasn't difficult for us to get down there and collect quite a few samples there.
Q. So looks like you collected a lot of samples on removal as well.
A. Yes, we did.

Q. We talked about OSHA's permissible exposure limit. Did OSHA also have a short-term exposure limit?
A. Yes, they did.
Q. During 1978 what was that limit?
A. That limit was ten fibers per CC.
Q. And we've indicated that on this graph; is that correct?
A. That's correct.
Q. And so these samples at the time of your study were well below that excursion limit?
A. That's right. And that's the appropriate limit that you would compare these standards to, these operations to because they're short-term operations. They're not long-term operations. You know, they're not -- it's not something that's done on an 8-hour basis other than the storage, for example.
Q. Did OSHA ultimately adopt a lower short-term exposure limit?
A. They did.
Q. And what was that?
A. It's currently one fiber per CC.
Q. And it's indicated here since 1988. To your knowledge, that hasn't been changed since 1988?
A. That's correct.
Q. Approximately 25 years. And so how do your samples compare with the OSHA short-term exposure limits?

A. They're lower than even the current short-term exposure limit.
Q. There's some photographs in your study; is that correct?
A. Yes, there are.
Q. I saw this photograph here, Figure 14. What was it in relation to?
A. That shows a flange where the gasket has been removed, but you can still see some of the gasket debris adhering to the flange. So that's an example of a flange where they have to do some clean up after the gasket has been removed.
Q. So the gasket has already been removed. They just have to clean up that debris.
A. That's correct.
Q. Those type of activities, what did you find?
A. Again, we found very low levels of exposure no matter which way they did those operations.
Q. Did you make recommendations in your report?
A. Yes, we did.
Q. I'd like to turn to those if we could. Is this a page from that -- from the recommendation section?
A. Yes.
Q. Oh, I'm sorry. This is the clean up following removal part, right. I want to ask you about this.
A. Yeah, that's not a recommendation. That summarizes the results we got from that particular operation.

Q. All right. And after removal of gaskets from an object, residual pieces of gasket material may have remained attached. Figure 14, that's the photo in the bottom there, shows residual material remaining on the flange.

And so it looks like hand scraping was the operation to remove this residual material.

A. That's correct.

Q. And it indicates that less than 0.05 was the range and the average for four samples. Can you tell us what does that mean, less than 0.05?

A. Well, it -- you can't -- as scientists we never report zero. You can never say that something is zero. But if we had found any fibers for these samples, .05 is the lowest we could have gone. So we don't know how much we had, but we know it was less than that.

It's like trying to weigh a letter on your bathroom scale. You can't say it weighs an ounce because your scale doesn't go that low. You can say it's less than a pound possibly. So that's the sort of thing we're doing here.

Q. So for cleaning up a flange face like Figure 14 using hand scraping, were there any controls?

A. No.

Q. None of the housekeeping that you were talking about before?

A. No.

Q. Now, let's look at the recommendations.
Hang on just one second.
THE COURT: Do you want to work on that and do it tomorrow?
MR. HARRIS: We certainly could take a break if you would like now, Your Honor.
THE COURT: All right. Why don't we go ahead and do that.
MR. HARRIS: I'll be happy to. I'll be ready to go in 45 seconds or 30 seconds.
THE COURT: Well, go ahead. We'll go for 15 more minutes.
MR. HARRIS: Okay.
BY MR. HARRIS:
Q. Okay. We're back in business.
Recommendations. The first section, it's entitled "Work practices and procedures for asbestos-containing gaskets;" is that correct?
A. Yes.
Q. It says, "The recommended work practices and procedures are based on reducing asbestos exposures to gasket workers to below 0.1 fibers per CC."
Now, why did you choose that?
A. We chose that because that was the level at which medical monitoring was to begin. We wanted to see if we could get

exposures below that.
Q. "The engineering controls for achieving this goal are feasible and the necessity for medical surveillance for large numbers of gasket workers is eliminated. If less stringent goals, such as 0.5 and 2.0 fibers per CC are acceptable, the work practices can be modified accordingly to summary table XII. The control measures are as follows."
And did you make recommendations for each of the operations that you studied?
A. Yes, we did.
Q. And so for secondary manufacturing, what those one or two shipyard workers were doing, cutting gaskets or fabricating gaskets all day, you actually recommended some controls; is that correct?
A. We did. For secondary manufacturing we recommended local exhaust ventilation and what we called a port vac which is a high efficiency vacuum cleaner with a HEPA filter on it to clean the areas.
Q. What is local exhaust ventilation?
A. It's a ventilation that draws the dust away from the source where it's generated.
Q. All right. For the end user activities, did you make other recommendations?
A. Yes.
Q. So for storage, any recommendations?

A. We didn't make any recommendations at all. We said it would be nice if you put it in a plastic bag.
Q. For hand shaping?
A. The same thing. We said that the waste should go in a plastic bag. And those were -- that wasn't to reduce exposure. It was because the navy had disposal requirements for asbestos-containing materials and so we wanted it already put in the plastic bag to make those easier.
Q. Installation: No special controls are necessary.
And then for removal and flange clean up following removal, what did you recommend?
A. The same recommendation as shaping. Just put the scrap in a plastic bag for disposal.
Q. And does that reduce the exposures from those operations?
A. It does not.
Q. So Mr. Liukonen, I want to go back to the slide I jumped up on a few minutes ago.
The committee's experts have lodged some criticisms of this study. You're familiar with those?
A. I am.
Q. You read their reports that they filed in this case?
A. Yes, I have.
Q. And you've read their depositions?
A. Yes.
Q. Okay. I want to ask you about some of the things that

they've claimed.

First, we've heard that they've said that it's not a real world study. Is it a real world study?

A. Absolutely. It was conducted in the shipyard under real world work conditions.

Q. Was it simulated in any way? We've heard about Dr. -- or we'll hear about Dr. Longo and how he's done some simulation studies. Were you simulating any activities here?

A. There were no simulations whatsoever. Whatever happened is what we tested.

Q. Is this a -- this wasn't a simulation study; it was a field study.

A. That's correct. It's a field study.

Q. Now, are there limitations to field studies?

A. Absolutely.

Q. What are those limitations?

A. The main limitation from a field study is that you can't control for other fibers from any other source so that you're not a hundred percent sure that the fibers you're measuring are from the gasket. They may be from an operation adjacent or in the next space over.

Q. Another criticism is that no samples were taken aboard ship. Is that true?

A. That is not true. Most of the end user activities were taken aboard ship.

Q. Actually, does the report address that?
A. It does. It specifically says they were taken aboard ship.
Q. This is Mr. Templin's report where he says, "A second naval study," referring to your study, "concerning gasket operations, this one conducted by the U.S. Navy and often referred to as the Bremerton study, was reported in May 1978. It was not, however, conducted in the field of workers under actual conditions as contended by Boelter," et al. Do you disagree with that?
A. Absolutely.
Q. Okay. On the samples aboard ship under project definition, there's a line here. Can you tell us what they say about where the samples were collected.
A. We said, "The majority of the samples were collected by NRMC Bremerton at Puget Sound Naval Shipyard in various shops and aboard ship."
Q. What about the end user activities, were those samples collected aboard ship or in shops or...
A. Virtually all, if not all, of those were collected in -- aboard ship.
Q. "No major overhauls were going on at the shipyard." I believe Mr. Beckett had said in his deposition maybe that all the operations involving gaskets couldn't be sampled because there were -- there wasn't a major overhaul going on in the

shipyard. Is that accurate?
A. That is not accurate. We had -- we had six dry docks. They were almost always full. Our largest dry dock, dry dock number 6, pretty much always had an aircraft carrier in there. And as this document indicates, I looked it up to verify, and indeed there was an aircraft carrier in the largest dry dock.
Q. In response to a FOIA request that Garlock's lawyers once made, they produced a list of all the operations, all the ships that came in through the Puget Sound shipyard over the course of 30, 40 years. You've seen that document?
A. I have.
Q. You produced it, I believe, at your deposition. And we've highlighted one of the ships that's listed there and the dates that it was in service. It says, "CV-43 Coral Sea."
Is that an aircraft carrier?
A. It is.
Q. It came in for service on March 7, 1978, and left February 7, 1979. How does that tell you that that's an overhaul?
A. Because of the length of time that it was there. It was there for nearly a year. So it was there for extensive work and the timing is absolutely perfect for our study.
Q. When were you assigned to do the study and when did you publish the study?
A. The original correspondence was in February of 1978. We

published it in May of 1978.
Q. And so this March 7, the first phase of overhaul is what?
A. It's absolutely perfect. That's when the work was being done.
Q. "Controlled environment." I believe Mr. Templin also said, "Rather, it was performed" -- referring to your study. "It was performed within carefully demarcated barricades placarded by large conspicuous asbestos dust hazard caution signs, by employees wearing respirators and protective clothing, and utilizing a variety of work practice, engineering, and housekeeping controls."
Now, what's he talking about there?
A. Well, that's incorrect. There are some pictures. This is an example. When we went to test, the person using the shear, he or his supervisor or someone had misinterpreted our instructions for working with friable insulation. Decided that those applied to gaskets, which they did not. But that's an example of what he did. He put on protective clothing. He put up a sign.
There you can see the rope and the sign. And had that been friable thermal insulation we were working on, according to our instructions you could work on this side of the sign without any problem.
Q. Okay. So back in 1978 at a shipyard, when you were talking about minimal controls to protect from potential

exposure to insulation, this was the rope and the sign that you were talking about.
A. That's the rope and the sign, yes.
Q. So in 1978 there's not the tent or the plastic sheeting that goes up to completely encase the area; is that correct?
A. That is correct. And when I arrive at a job site like this, if someone wants to use more precautions than are required, that's fine with me. I never tell them to use less precautions.
Q. What operation is this?
A. This is machine shearing.
Q. Is this one of those secondary manufacturing operations or is this an end user operation?
A. This is one of those secondary manufacturing operations.
Q. And this is one of the two people in the shipyard that did that job?
A. This is kind of like two and a half. This job is done at times so this is -- this is a little bit more than two. This is a third person that does some of it.
Q. All right. Here is another picture out in the shipyard. Looks like he may actually be working on Garlock gaskets there; is that correct?
A. That's correct. That's the same individual and using the same precautions. And this machine is called a nibbler. I think some people call it a fly cutter.

Q. Is this a secondary operation activity or an end user activity?
A. It's a secondary manufacturing operation.
Q. All right. And so this is the work that was going on in shops that was referenced at the beginning of the report?
A. That's correct.
Q. And so for the end user activities that were going on aboard ship, you don't have photographs of people actually working on ships; is that correct?
A. That's correct, none of our photographs were aboard ship, I don't believe.
Q. Why would that be?
A. The shipyard was very tightly controlled on that sort of issue and they didn't even allow their own photographers to go take pictures aboard ship at this time.
Q. Okay. Now, Dr. Longo has made an interesting criticism. The committee's expert, Mr. Beckett, evidently went down to Dr. Longo's lab and had a conversation with him. And Dr. Longo recants that in his report.
He says, "Bremerton's gasket study could not be used as an indicator on all potential asbestos exposure removal projects at Bremerton. Mr. Beckett said this was especially true of the Bremerton study since it was done under abnormal conditions because of the presence of a number of senior personnel who were observing the study, a shipyard

photographer documenting the study, and housekeeping controls utilized during the study."

And then he said in another deposition, "As Mr. Beckett stated, this was a study that was -- where you had ship photographers, you had all the brass watching the studies, and they were not representative of what usually goes on with gasket studies."

Now, you've read Mr. Beckett's deposition, correct?

A. I have.

Q. Do you recall what he said about that?

A. He said he never said that.

Q. Right.

A. Which is absolutely true. There were no navy brass present during the sampling. There were industrial hygienists or industrial hygiene technicians present during the sampling. There were occasionally a shipyard -- or was occasionally a shipyard photographer present to take some of these pictures, but there were no navy brass present.

Q. And if navy brass were present or somebody was influencing the results of your study, would that be something that would be noted?

A. Absolutely. If we thought it wasn't representative of real world work conditions, we would have noted it.

Q. And the purpose of -- one of the purposes of the study was to determine whether people working with gaskets actually

needed to be medically monitored, correct?
A. That's correct.
Q. And if people were influencing the results of the study, would you have to note that?
A. Absolutely.
Q. Mr. Liukonen, you worked in that shipyard for three years; is that correct?
A. Four years.
Q. Four years. And you knew people that worked there.
A. Yes, I did. Bremerton is a small town.
Q. And did you know people that were actually working in the shipyard that were working with gaskets?
A. Yes, I did.
Q. If you thought that they needed controls when they were working with gaskets out in the field, would you have made recommendations for controls?
A. Absolutely.
MR. HARRIS: Your Honor, would this be a good time to take a break?
THE COURT: It's 5:30. Let's take a break and we'll come back at 9:30 tomorrow morning.
(Evening recess at 5:29 p.m.)
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UNITED STATES DISTRICT COURT

WESTERN DISTRICT OF NORTH CAROLINA

CERTIFICATE OF REPORTER

I certify that the foregoing transcript is a true and correct transcript from the record of proceedings in the above-entitled matter.

Dated this 24th day of July 2013.

s/Cheryl A. Nuccio
Cheryl A. Nuccio, RMR-CRR
Official Court Reporter